

Nicolas Hardt
Johannes Kuttenger

Craniofacial Trauma

Diagnosis and Management

 Springer

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Nicolas Hardt
Johannes Kутtenberger

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Diagnosis and Management

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Dedication

*Dedicated to the excellent surgeon, mentor, and friend
Prof. Dr. Dr. Emil. W. Steinhäuser
Director emeritus Clinic and Policlinic of Oral and
Cranio-Maxillofacial Surgery
University Clinics
Friedrich-Alexander-University, Erlangen-Nuremberg, Germany*

and

*Anna-Marie Hardt-Oswald
for her inspiration and brilliant encouragement*

Prof. Dr. Dr. Nicolas Hardt

Citation

“The more severe type of injury involving the middle third of the facial skeleton results in a characteristic appearance with “ballooning” of the soft tissues, particularly the eyelids, accompanied by subconjunctival and circumorbital ecchymosis and often cerebrospinal fluid rhinorrhoea. The posteriorly and downwardly displaced upper jaw causes gagging of the occlusion on the posterior teeth. The overlying edema renders accurate diagnosis of the underlying skeletal injuries difficult and it is essential to possess a thorough knowledge of the anatomy, physiology and pathology of this region if these injuries are to be treated intelligently and successfully”

(Rowe and Killey 1968, *Fractures of the Facial Skeleton*)

Preface

Facial fractures, particularly those resulting from severe injuries with multiple fractures in the cranio-maxillo-facial region, are the most common form of neurocranial injuries (Hausamen and Schmidseider 1975; Machtens 1987; Brachvogel et al. 1991; Wahlmann and Wagner 1991).

Depending on the complexity and level of the fracture, the frontobasal involvement of all craniofacial injuries varies between 30 and 70% (Manson et al. 1987; Raveh et al. 1992; Weerda 1995; Joss et al. 2001). In addition to the challenging reconstruction of severe craniofacial injuries, specific diagnostic, pathogenetic and therapeutic problems arise as a consequence of the accompanying frontobasal fractures.

A considerable optimization in the treatment of these profound injuries can be achieved by a routine team approach of maxillofacial, neurosurgical, and anesthesiological specialists with the appropriate diagnostic and therapeutic resources at their disposal. The interdisciplinary treatment of patients with severe craniofacial injuries is state-of-the-art and focused on trauma centers equipped with the adequate infrastructure (Reulen and Steiger 1994; Weingart et al. 1996; Seidl et al. 1998).

The purpose of the present monograph is to analyze and introduce an established therapy concept for craniofacial fractures with anterior subcranial involvement, with reference to the surgical approach and the postoperative results. Of particular interest are etiological, epidemiological, and pathomechanical characteristics in neurocraniofacial injuries.

The monograph is based on the analysis of a documented collective of 268 severe craniofacial injuries in the context of 18,456 maxillo-facial injuries treated with assured data regarding quantity and quality in relation to the extent and pattern of injury, epidemiology, and reconstructive procedures in the varying fracture compartments, including perioperative management.

Furthermore, surgical indication, time of intervention, and the maxillofacial-neurosurgical treatment modalities for the subcranial, craniofrontal, craniofacial, and frontobasal regions are looked at in detail, as well as the principles of reconstructing the cranio- and maxillofacial skeleton.

The editors hope that this manual will be an indispensable reference for residents in maxillofacial training and attending cranio-maxillofacial and for neurosurgeons in the highly specialized field of cranio-maxillofacial neurotraumatology.

Luzern, Switzerland

Nicolas Hardt
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Classification and Diagnosis

1.1 Anterior Skull Base

The anterior cranial fossa is of vital importance in traumatology of the craniofacial region (Manson 1986). In close proximity there are connections to the neighboring cerebral regions, to the olfactory bulb and tract, to the frontal cerebral lobe, to the anterior temporal lobe, to the pituitary gland, and to the brainstem (Fahlbusch and Buchfelder 2000).

From anterior to posterior, the skull base is divided into the anterior, middle, and posterior cranial fossa. The inner surface of the anterior cranial fossa is composed of the ethmoidal, frontal, and sphenoidal bones (Lang 1985, 1987, 1988, 1998; Lang and Haas 1979; Schiebler and Schmidt 1991). Topographically, the anterior cranial fossa is subdivided into a medial section (lamina cribrosa and crista galli), a lateral section (orbital roof/ethmoid/posterior wall of the sinus), and a posterior section (sphenoid/sella) (Fig. 1.1).

The most important transverse interfaces are the sutura fronto-ethmoidalis and the sutura sphe-no-ethmoidalis. The sutura sphe-no-ethmoidalis forms the anatomical border between the ethmoidal bone and the lesser wing of the sphenoid bone, which, in turn, marks the border to the middle cranial fossa.

This line borders on the lesser wing of the sphenoid bone and the connecting line between the optical foramina. The canals for both optical nerves are situated here, anterior to the hypophyseal fossa. The sphenoidal plane belongs to the anterior cranial fossa.

1.1.1 Cribriform Plate/Crista Galli

The ethmoidal bone lies prominently appendant to the paired cribriform plate and crista galli in the medial section of the floor of the anterior cranial fossa.

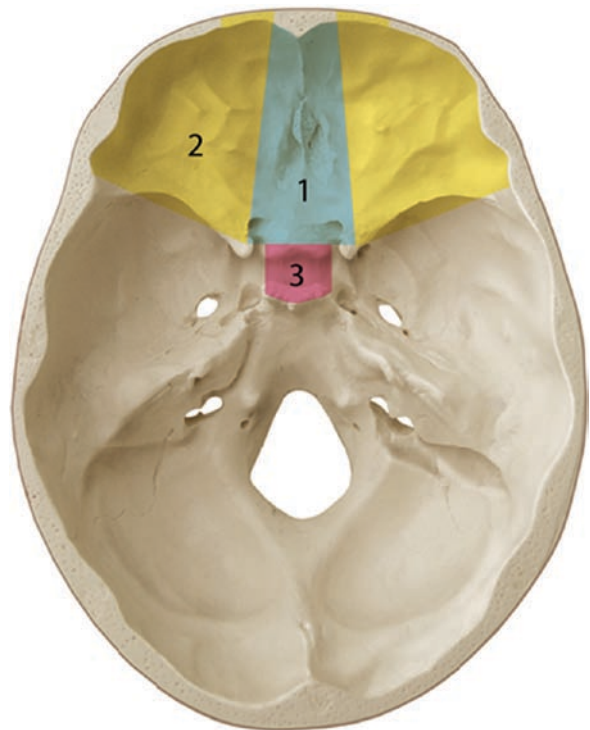


Fig. 1.1 Topographical subdivision of the anterior cranial fossa into a medial section (1 lamina cribrosa/crista galli), a lateral section (2 orbital roof/ethmoid/posterior wall of the sinus) and a posterior section (3 sphenoid/sella)

The cribriform plate itself is thicker than the roof of the ethmoid bone. The falx cerebri, which separates the two cerebral hemispheres, is almost anchored onto the crista galli, a usually pneumatized bony ridge rising sagittally in the middle of the cribriform plate. The foramen caecum lies in front of the crista galli and is surrounded by the ethmoidal bone posteriorly and laterally and anteriorly by the frontal bone (Rohen and Yokochi 1982; Vesper et al. 1998).

The cribriform plate is a perforated strip of bone. Between 26 and 71 foramina, on average 44, can be

found on both sides of the midline, through which the olfactory fibers pass, enclosed in the dural sheath and the subdural space, as they lead to the olfactory bulb (Schmidt 1974; Lang 1998).

Lateral to the cribriform plate, the orbital parts of the frontal bone constitute the greater part of the orbital roofs and the floor of the anterior cranial fossa. The sphenoidal plane forms the posterior border of the cribriform plate (Lang 1998). Normally, the sphenoidal plane slightly overlaps the posterior margin of the cribriform plate.

As a rule, the posterior ethmoidal artery and its fine collateral nerve enter the region in the lateral section of this overlap (Krmptocic-Nemancic et al. 1995).

In the anterior sector of the cribriform plate there is a large, elongated aperture (foramen cribro-ethmoidale), through which the thickest branches of the anterior ethmoidal nerve and artery pass on their way to the nasal cavity (Jackson et al. 1998; Donald 1998) (Fig. 1.2).

1.1.2 Fossa Olfactoria

The olfactory fossa meets the medial and upper paranasal walls laterally. As a rule, the major part of the upper wall is formed by the orbital section of the frontal bone. Seldomly, the ethmoidal bone is also involved in forming the upper wall of the ethmoidal cells (Lang 1988).

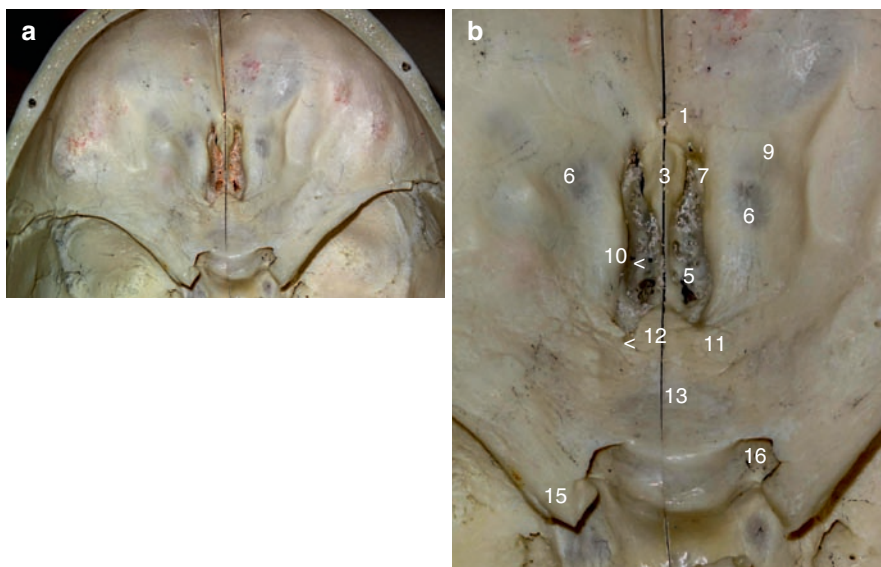
Concurrently, the exceptionally thin ascending osseous lamellae of the cribriform plate form the medial part of the ethmoidal labyrinth. The deeper the lamina cribrosa lies, the higher are the ascending lamellae (Fig. 1.3).

On average, the olfactory fossa is approximately 15.9-mm long and 3.8-mm wide (Lang 1988). Keros (1962) determined the depth of the olfactory fossa to be 5.8 mm anteriorly and 4.8 mm in the posterior third. The distance between the lamina cribrosa and the highest point in the ethmoidal labyrinth measures 6.9 mm in the anterior third and 5.8 mm in the posterior third. The lowest inner cranial point of the lamina cribrosa lies approximately 7.9 mm below the nasion on both sides (Lang 1987, 1998; Krmptocic-Nemancic et al. 1995).

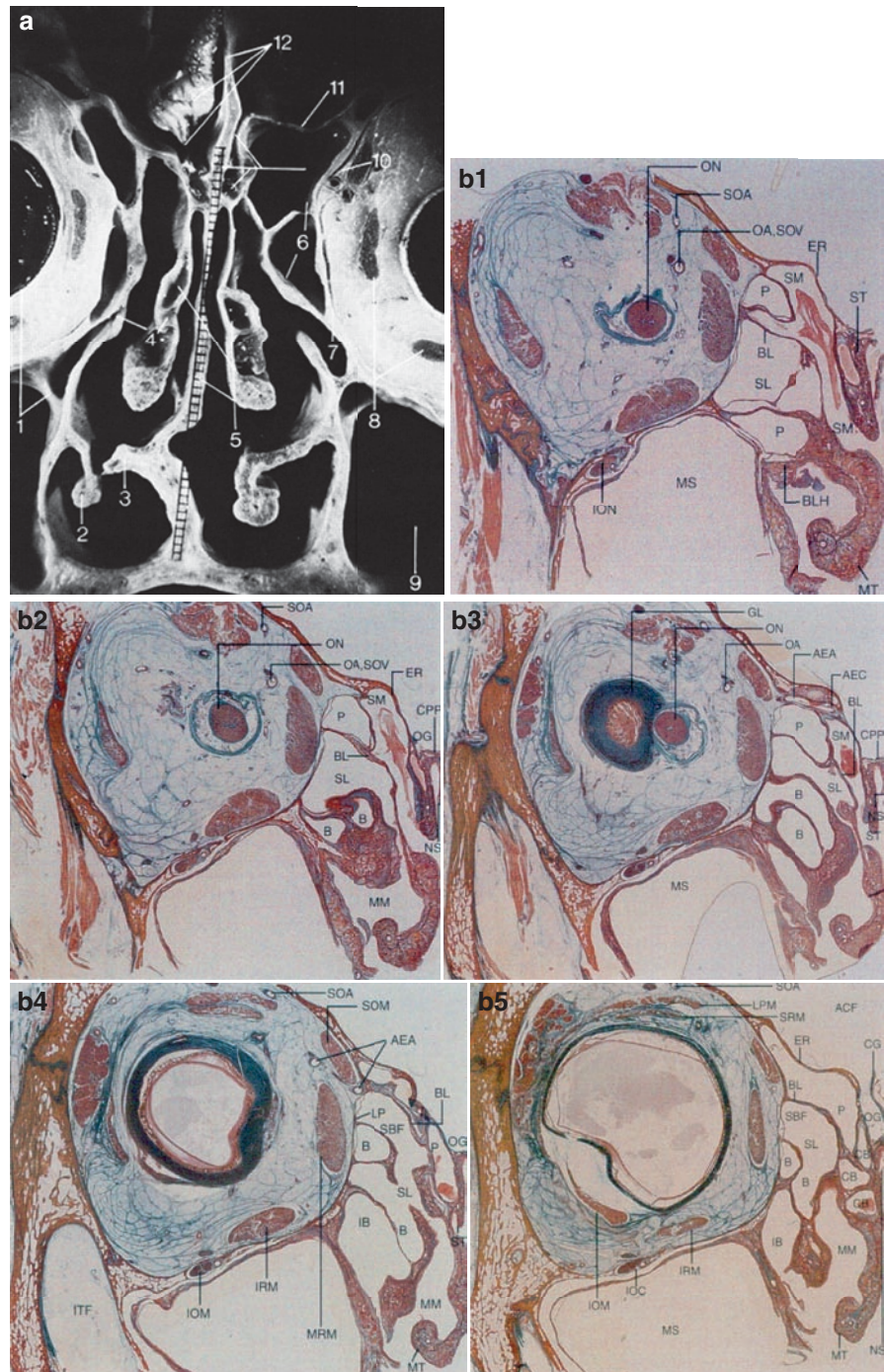
Keros (1962) accounted for a shallow fossa (1–3 mm deep) in 12%, a fossa with average depth (2–7 mm) in 70% and a deep fossa (8–16 mm) in 18%. These differences in niveau can also be described as “*encaissement des ethmoids*” (Probst 1971).

- The deeper the cribriform plate lies in relation to the ethmoidal roof, the wider is the very fine ascending osseous lamella (os frontale) between the cribriform plate and the roof of the ethmoid. In viscerocranial injuries, the difference in height between the lamina cribrosa and the residual base predisposes fracturing in the thin lamina with an imminent danger of dural injury (Sakas et al. 1998) (Fig. 1.4).

Fig. 1.2 Anterior cranial base with cribriform plate and penetrating medial and lateral olfactory foramina (after Lang 1983a, b, 1998). 1: Foramen caecum 3 Crista galli, 5 Cribriform plate, 6 Roof of the anterior superior ethmoidal cells, 7 Ethmoidal sulcus, 9 Fronto-ethmoidal suture, 10 Lateral lamina of the lamina cribrosa, 11 Os frontale 12: Spheno-ethmoidal suture / Planum sphenoidale 13: Sphenoidal limbus 15: Anterior clinoid process 16: Optic channel



B: ethmoidal cells
LP: lamina papyracea
ER: ethmoidal roof
ST: superior turbinate
ACF: anterior cranial fossa
OG: olfactory fossa
CG: crista galli
AEA: ante ethmoidal artery
CCP cribriform plate
ON: optic nerve



(Continued)

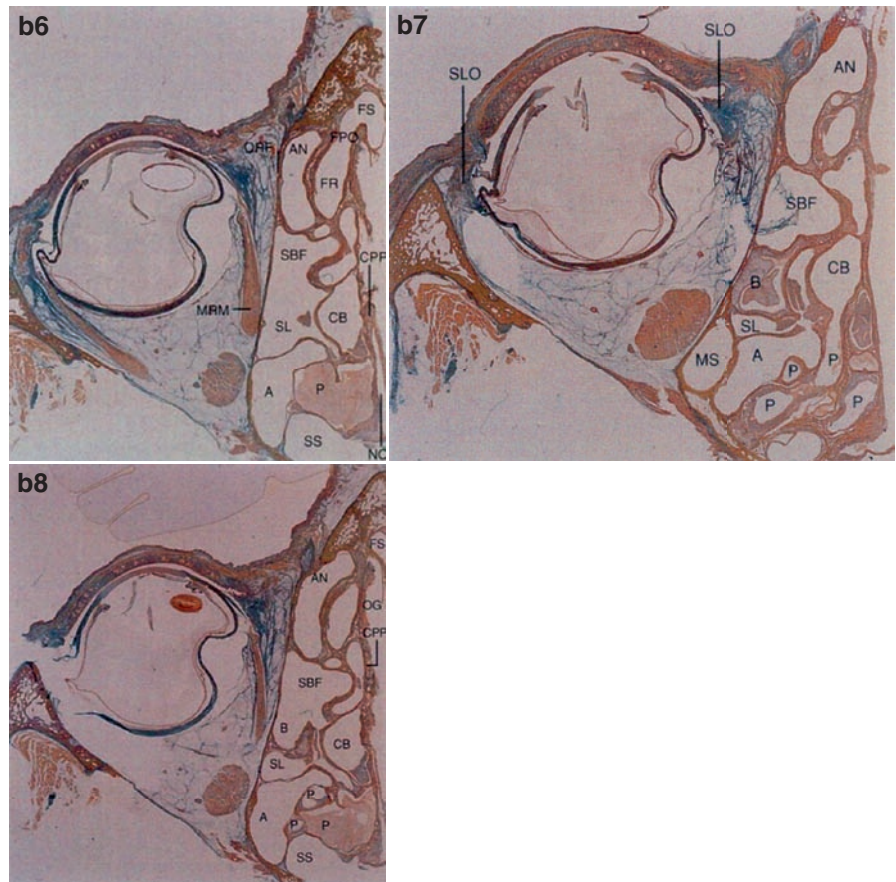
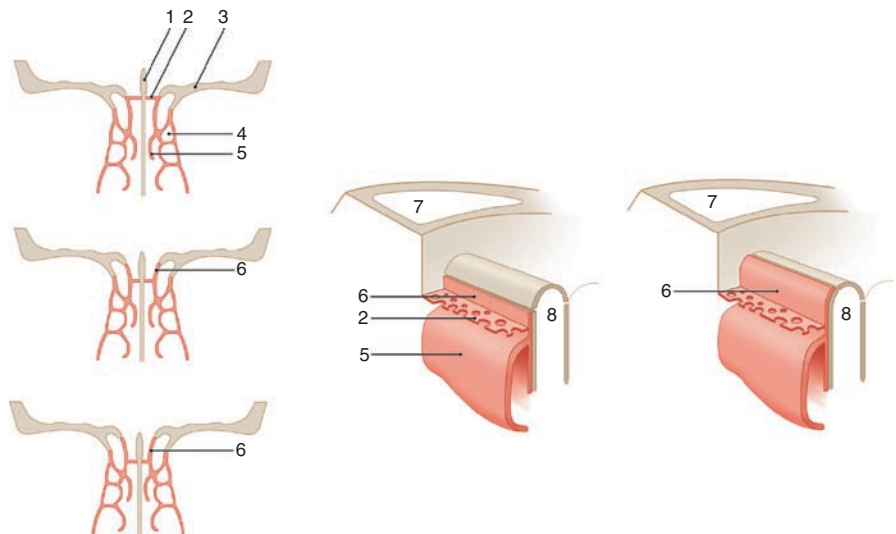
Fig. 1.3 (continued)

Fig. 1.4 Variable position of the cribriform plate in comparison with the roof of the ethmoid, caused by the different extension of the lateral lamina of the cribriform plate (mod. a. Probst 1971, Krmptovic-Nemancic et al. 1995). 1 Crista galli, 2 Cribriform plate, 3 Os frontale, 4 Ethmoid, 5 Concha medialis, 6 Lateral lamina, 7 Sinus frontalis, 8 Sinus ethmoidalis



1.1.3 Roof of the Orbit

The orbital roof of the frontal bone exhibits a filmy osseous structure displaying impressions and ridges. The digital impressions and crest-like jugae form an irregular relief with hills and valleys in the region of the orbital and ethmoidal roofs. The prominent bone is substantially thicker than that in the depressed zones (Probst 1986). Here there is a major site of predilection for base fractures and dural injuries and, consequently, also a primary site for liquor fistulae (Probst and Tomaschett 1990).

1.1.4 Dura

Aberrant to other regions of the cranial skeleton and skull base, the frontobasal region displays anatomical anomalies in the configuration of an osseous cranial vault with depressions, ridges, and septa. The association to the dura mater padding is closer in the frontobasal region than the remaining skull interior. The dura itself is comparatively thin and particularly tightly anchored to the bone along the sutures and foramina. There is an exceptionally strong fixation of the dura to the cribriform plate, roof of the labyrinth and crista galli. The epidural translational displacement layer, as found in the middle and posterior cranial fossa, is missing here (Vajda et al. 1987). Furthermore, in the region of the foramina the dura is attached to the sheath of the first cranial nerve. It is histologically proven that the subarachnoid space occasionally extends caudally along the olfactory fila, through the cavities of the cribriform plate (Probst 1971).

- In the region of the olfactory foramen, the virtual dural cover is lacking and there is a mere arachnoidal covering; so, in the case of fracture, liquor fistulas may easily occur (Samii et al. 1989; Okada et al. 1991; Sakas et al. 1998).

1.1.5 Arterial Supply: Skull Base/Dura

The floor of the anterior cranial fossa and the dura mater are supplied by the anterior ethmoidal artery, whose branches ascend into the falx cerebri, forming the arteria falcea anterior, and pass through the

foramina cribrosa on their way to the medial and lateral walls of the nasal cavity (Lang 1998).

Branches of the arteria carotis interna and arteria cerebri anterior may be involved in supplying the far-most medial floor regions of the anterior cranial fossa.

The lateral floor region of the anterior cranial fossa gets its supply from the frontal ramus of the middle meningeal artery, whose meningo-orbital branch penetrates the floor of the anterior cranial fossa and anastomoses with the rami of the ophthalmic artery (Lang 1998).

1.2 Paranasal Sinuses

From an evolutionary point of view, the paranasal sinuses are convexities of the nasal cavity into the neighbouring bone. Their mucosa are a continuation of the nasal mucosa; thus, a close relation exists between the varying paranasal sinuses. They are very variable with regards to dimension and shape (Lang 1985, 1998) (Fig. 1.5).

1.2.1 Frontal Sinus

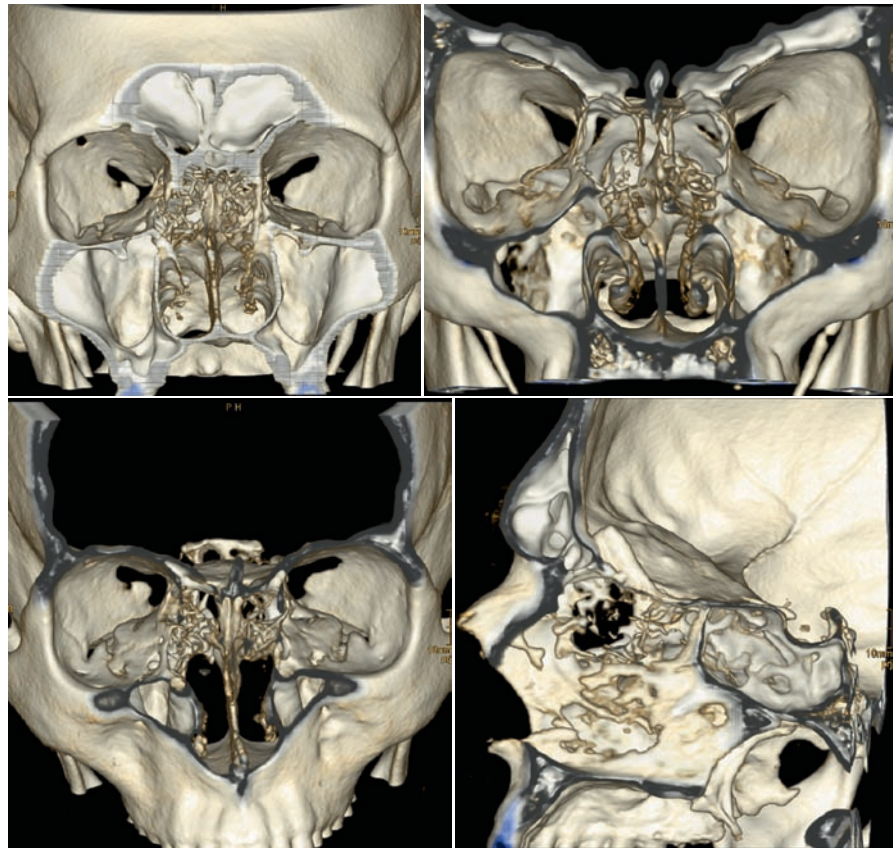
Dimension and form of the frontal sinuses vary greatly. They may be totally absent (aplasia) or extend asymmetrically into the orbital roof. In the latter case, they may even reach the anterior margin of the lesser wing of the sphenoid bone. Laterally, the frontal sinus can extend as far as the zygomatic process of the frontal bone and occasionally comprise the lateral orbital wall. The roof of the frontal sinus partially constitutes the floor of the anterior cranial fossa.

The extent of the frontal sinus in the orbital roof section of the frontal bone is particularly important during surgery, when approaching the orbit from the anterior cranial fossa (Lang 1998).

1.2.2 Ethmoid

The ethmoidal labyrinth – in the center of the facial skeleton, with proximate anatomical connections to

Fig. 1.5 Radiological coronal, semicoronal and sagittal CT sections through the orbit, the ethmoid complex, the sinuses and the anterior base. Note the important subcranial position of the ethmoido-sphenoidal complex in the centre of the midface



the orbit, nose, and residual paranasal sinuses, and also situated in the anterior cranial fossa – has exceedingly great significance as:

- A link between the viscero- and neurocranium
- A central midfacial component
- The ontogenetic origin of the paranasal sinus system
- The site of olfactory cognition

The ethmoid measures 3–4 cm in length, 2–2.5 cm in height and 0, 0.5–1.5 cm in width (Lang 1987). The ethmoidal cells border medially on the nasal cavity, caudally on the maxillary sinus, and cranially on the anterior cranial fossa, respectively the frontal sinus. The orbital boundary is formed anteriorly by the lacrimal bone, posteriorly by the papyraceous lamina of the ethmoid and caudally by the maxillary complex. The sphenoid is attached posteriorly.

As a rule, the adjacent medial and anterior regions of the orbital roof are pneumatized by the frontal sinus extensions (Kastenbauer and Tardy 1995) (Fig. 1.6).

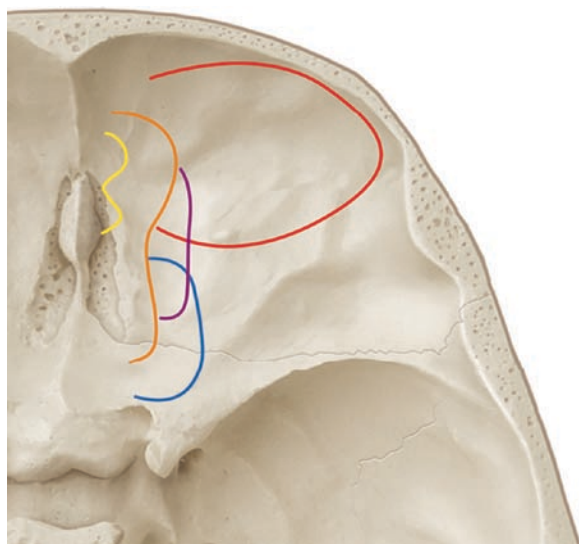
The ethmoid labyrinth is composed of a system of partially disjoined chambers, which one can divide

into an anterior and posterior ethmoidal cell system according to their position (Anon et al. 1996).

The horizontal lamella of the middle nasal concha forms the border. Genesis and anatomy of the anterior ethmoidal cells are constitutionally (fetal period) more complex than that of the posterior cell group.

The anterior ethmoidal cells drain into the hiatus semilunaris in the middle nasal meatus, the posterior ethmoidal cell system into the superior nasal meatus.

The posterior ethmoidal cells are located dorsal to the basal appendage of the middle nasal concha and ventral to the sphenoidal sinus. They are usually composed of three to four larger cellular cavities without having any complex anatomical connection to other paranasal sinuses. The posterior ethmoidal cells may extend as far as the ventral wall of the sphenoid sinus and laterally as far as the cavernous sinus. Occasionally they may even extend as far as the optical canal and the middle cranial fossa. A frontal bulla may protrude into the dorsal wall of the frontal sinus, where it is separated from the orbital cavity by a thin osseous lamella (Krmptovic-Nemancic et al. 1995).



- Minimal extension of the ethmoidal cells
- Mean extension of the ethmoidal cells
- Maximal extension of the anterior superior ethmoidal cells
- Maximal extension of the medial superior ethmoidal cells
- Maximal extension of the posterior superior ethmoidal cells

Fig. 1.6 Variability of the sagittal and transversal extension of the ethmoidal cells in the floor of the anterior cranial fossa (mod. a. Lang 1983a, b, 1987, 1998)

1.2.3 Sphenoid

The sphenoid bone forms the posterior connection between the mid-facial skeleton and the cranial base, whereas the ethmoid bone, which has a delicate honey-combed structure, forms the anterior connection. Antero-laterally the sphenoid connects to the zygomatic bone and antero-inferiorly via the pterygoid process it connects to the pyramidal process of the palatine bone.

The sphenoid sinuses border on the anterior, middle, and posterior cranial fossa, as well as on the sella turcica. The optic nerve passes through the lateral wall of the sphenoid sinus. It lies in close proximity to the internal carotid artery, the cavernous sinus and the cerebral nerves II-VI, as well as the sphenopalatine artery in the anterior sphenoid wall (Levine and May 1993; Messerklinger and Naumann 1995) (Fig. 1.7).

- A traumatic impact on the face can cause dislocated fractures in the frontobasal pneumatic cavities, which, in turn, may lead to disruptions of the inter-

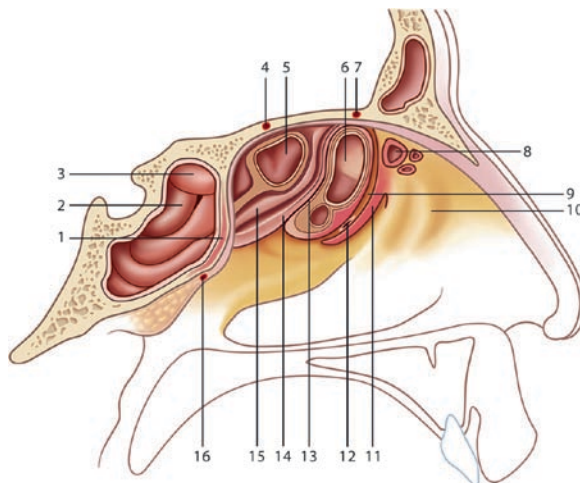


Fig. 1.7 Parasagittal section of the lateral nasal wall with the sub-basal ethmoidal-sphenoidal complex and its relationship to the frontal sinus and frontal base. The internal carotid artery and the optic nerve are prominent structures in the lateral wall of the sphenoid sinus. Note the relationship of the sphenopalatine artery to the inferior aspect of frontal wall of the sphenoid sinus (mod. a. Levine and May 1993) 1. Face of sphenoid 2. Internal carotid art. 3. Optic nerve 4. Posterior ethmoid art. 5. Air cell 6. Lamina papyracea viewed through ethmoid bulla 7. Anterior ethmoid art. 8. Agger nasi 9. Infundibulum 10. Lacrimal sac prominence 11. Uncinate process 12. Maxillary ostium 13. Sinus lateralis 14. Basal lamella 15. Lamella of superior turbinate 16. Sphenopalatine art.

nal mucous membranes, neighboring vital structures and dural injuries, so risking ascending intracranial infections (Boenninghaus 1971; 1983 Helms and Geyer; Theissing 1996).

1.3 Midface Skeleton

The central midfacial block, comprising the maxilla and the orbito-naso-ethmoidal region, constitutes the important osseous facial architecture. It incorporates the anterior skull base with the occlusal-mandibular complex and so predefines the vertical facial height. In the transverse plane, it combines both zygomatic-orbital regions, so determining the facial width (Maisel 1984; Jackson et al. 1986; Manson et al. 1987).

The midface – conceptually designed as a biomechanical light-weight structure with thin walled cavities – is subject to specific construction principles.

- It is composed of osseous cavities and forceful trajectories, which in turn convey great, static, compressive forces to the stabile skull base. Force dispersion occurs via prominent vertical,

horizontal, and sagittal osseous trajectories (Rowe and Killey 1968; 1970; Haskell 1985; Ewers et al. 1995).

The three vertical midfacial trajectories are: the anterior naso-maxillary pillar, the mid-zygomatic-maxillary pillar, and the posterior pterygo-maxillary pillar.

- The naso-maxillary abutment: runs as naso-frontal pillar from the canine tooth region, adjacent to the anterior bony aperture of the nose, through the frontal process of the maxilla to the upper orbital border and naso-ethmoidal region as far as the glabella region of the frontal bone
- The zygomatico-maxillary abutment – the middle trajectory: protracts as the zygomatic-maxillary pillar vertically above the zygomatic bone to the fronto-zygomatic sutures, to the frontal bone and via the zygomatic bone and arches into the temporal region
- The pterygo-maxillary abutment: runs posteriorly along the dorsal maxilla and the pterygoid of the skull base to the sphenoid bone (Fig. 1.8)

The midface is stabilized *horizontally* by a lower horizontal pillar composing the alveolar process and an upper *fronto-facial* pillar formed by the fronto-cranial compartment as well as a middle *infraorbital-zygomatico-temporal* pillar (Rowe and Williams 1985).

One can observe that no *sagittal* columns exist between the palate and the upper frontal arch (Mc Mahon et al. 2003). The upper orbital-interorbital midface complex is stabilized by two horizontal and four vertical latticed pillars (Mathog et al. 1995) (Fig. 1.9).

- This anatomical construction is of relevance when considering injuries to the central and lateral midfacial region. As a result of its special construction, the comparatively thin-walled midfacial compartment can absorb intense kinetic energy, so reducing the injury to the neurocranium in craniofacial injuries.

Principally, the midface only exhibits strong resistance against vertically applied forces. Although there is a lesser resistance against antero-posteriorly applied forces, it is combined with a high structural absorption capacity.

There is a 45° angle between the stabile *skull base* and the palato-occlusal plane. In contrast to the midface, this inclination results in a high resistance against an antero-posterior compression.

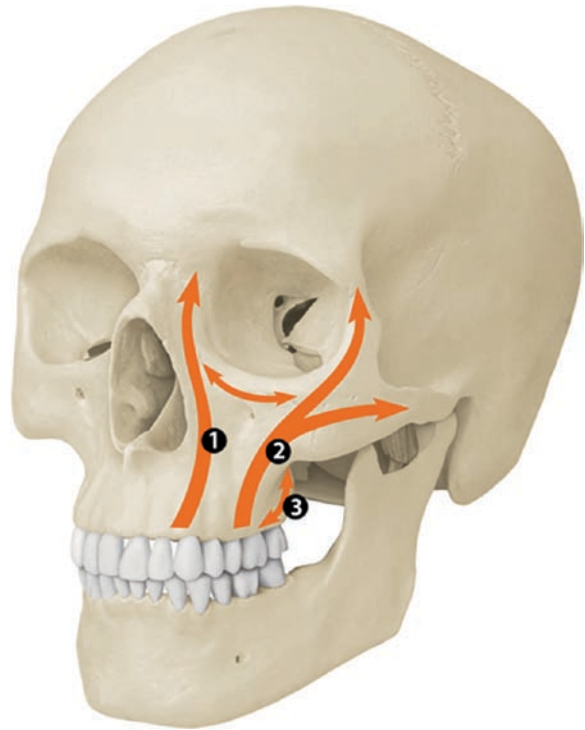


Fig. 1.8 Diagram of the vertical maxillary buttresses of the midface. These buttresses (bone-trajectories) represent regions of thicker bone, which provide support for the maxilla in the vertical dimension (mod. a. Prein et al. 1998). 1 Anterior medial naso-maxillary buttress, 2 lateral zygomatico-maxillary buttress, 3 posterior pterygo-maxillary buttress

In the case of an anterior-posteriorly applied force, the midfacial complex is driven against the sphenoid body, in such a way that the midfacial complex is dislocated en bloc posteriorly and caudally. This results in comminuted midface fractures with a typical dish-face deformity.

This also applies to assaulting forces to the midface from an antero-superior or lateral direction, which may cause the entire midfacial complex to shear off transversally from the cranial base (Rowe and Williams 1985) and induce subbasal avulsion fractures in the midfacial region including:

- Greater wing of the sphenoid bone
- Alar processes
- Ethmoid complex
- Frontal sinus

Collateral skull base injuries can be seen within the fracture compartment.

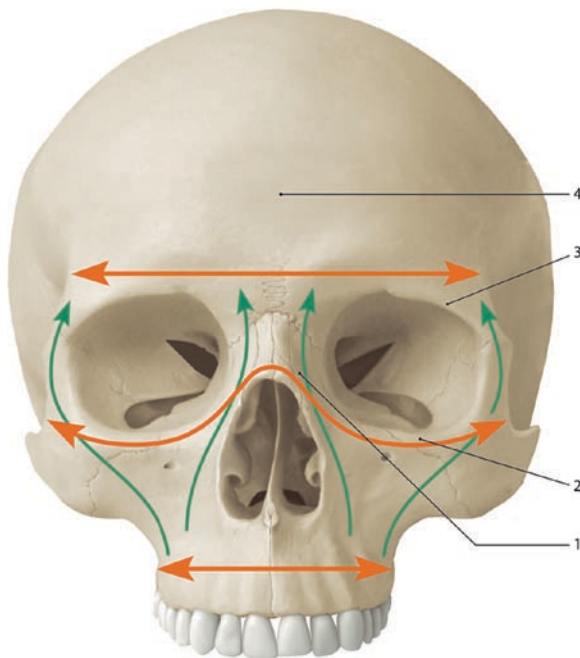


Fig. 1.9 Diagram of the transversal buttresses of the midface, represented by the horizontal supraorbital frontal bar, the infraorbital rims and the maxillary alveolar process. The superior orbital-interorbital complex (upper midface) is like a framework, stabilized by two horizontal and four vertical buttresses to which the more delicate facial bones are attached (mod. a. Mathog et al. 1995). 1 Frontal process of maxilla, 2 inferior orbital rim, 3 superior orbital rim, 4 frontal bone; orange lines transverse supraorbital-frontal, infraorbita, and alveolar buttresses, green lines vertical lateral zygomatico-maxillary and anterior medial naso-maxillary buttresses

1.4 Subcranial and Midface Skeleton

The midface and the anterior base of the skull form a structural and biomechanical entity. Whilst *anatomically* the midface ends subbasally, on the basis of the close topographical-anatomical, functional, and biomechanical complexity of both structures, the frontofacial and frontobasal regions are still considered as belonging to the midface (Ewers et al. 1995; Hausamen et al. 1995).

- The high incidence of combined midfacial-frontobasal fractures is based on the close topographical and biomechanical relationship of the osseous structures in the viscerocranium and skull base.

The *anatomical connection* between the midface and neurocranium is formed by the maxilla, the naso-ethmoidal complex, the palatal and vomerine bone, and the pterygoid process of the sphenoid.

- Through the pterygopalatal column of the sphenoid, the lower surface of the skull base is directly involved in constituting the posterior midface. Whereas through the frontal maxillary process, the naso-ethmoidal complex is indirectly involved in the anterior midfacial composition.

Biomechanically, both compartments – midface and neurocranium – are intimately connected through the intersection of the external as well as internal vertical and transversal facial trajectories. The horizontal as well as sagittal trajectories of the fronto-facial region are connected with the anterior skull base. Hence, in the case of *craniofacial fractures*, the frontobasal and frontofacial regions may be involved in complex severe facial injuries (Hardt et al. 1990; Ewers et al. 1995). This explains the high incidence of skull base fractures in midface fractures (Fig. 1.10).

Vertical forces along the naso-maxillary and zygomatic-maxillary pillars are indirectly absorbed by the frontal bone, whilst the posterior vertical forces along the pterygo-maxillary column may be directly transferred to the skull base. Sagittal forces are conveyed to the temporal bone via the zygomatic-temporal column.

Forces applied to the central maxillofacial region may cause fractures on the subbasal level, which in turn may traumatize neighboring vital structures. The frontal skullbase and dura are in immanent danger. Acute and chronic ascending intracranial infection may result (Helms and Geyer 1983).

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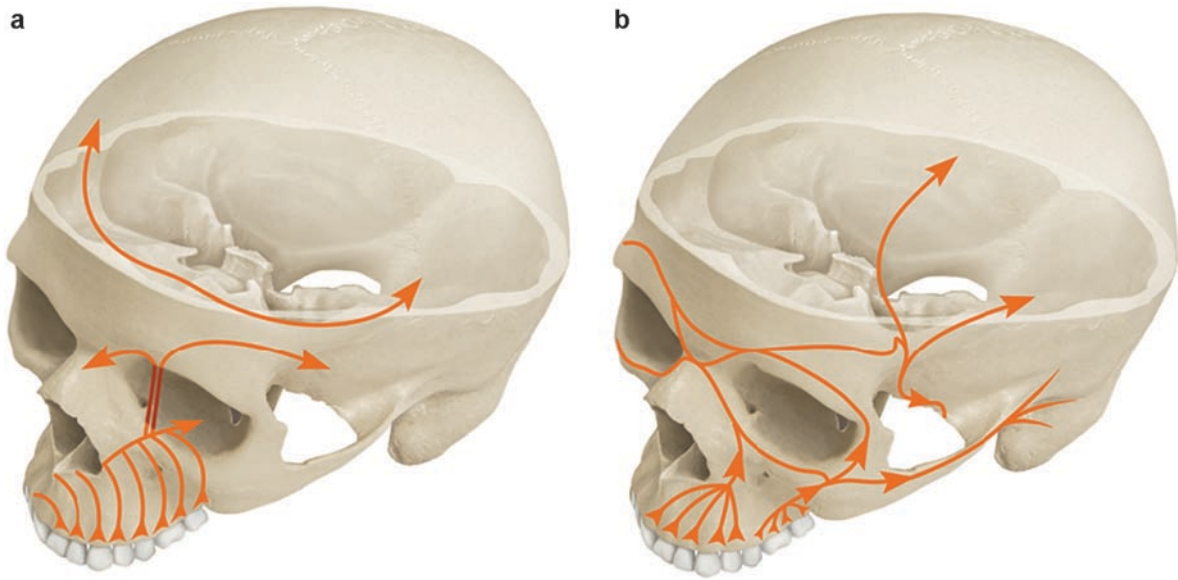


Fig. 1.10 Relationship between the bone trajectories of the midface and the anterior cranial base (mod. a. Ewers et al. 1995). (a) Internal transversal trajectories. (b) External vertical and transversal trajectories

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Until a few years ago, conventional X-rays were the imaging standard for cranio-cerebral and facial trauma. Today, however, computed tomography (CT) has become the primary imaging method, along with significant technical improvements, especially with the development of multislice CT.

Conventional X-rays are relatively sensitive to cranial vault fractures, but insensitive to fractures of the skull base and facial skeleton. CT enables a precise diagnosis of all kind of fractures of the facial skeleton and skull base, and additionally delivers information about intracranial bleeding and injuries to the cerebrum. In the multi-traumatized patient, CT can be extended to the cervical spine as well as the trunk if necessary. A complete body check for traumatic lesions can be done within a few minutes, including the brain, spine, bone, and organs. Thus, conventional X-rays of the skull are no longer used in the case of head traumas or polytraumatized patients; CT is widely accepted as the primary imaging method of choice. Nevertheless, the following provides an overview of all imaging methods, including conventional X-rays.

2.1 Conventional X-Rays

The standard X-ray exposures for the skull are summarized in Table 2.1. Standard projections are the anterior/posterior (AP) and the lateral view of the whole skull. These images are sensitive to skull fractures, which fall under two general categories: (1) direct fractures identifiable as fracture lines, fracture gaps and dislocation of

osseous fragments of the skull; (2) indirect fractures identified as opacification of the paranasal sinuses and soft tissue emphysema. For the facial skeleton, the semi-axial view of the midface is required either in occipito-mental or occipito-frontal projections, while fractures of the mandible require the panoramic and the Clementschitsch view.

The sensitivity of the different exposures for fractures varies depending on fracture type. Some simple fractures can be well displayed on dedicated X-ray projections. On the other hand, complex fractures can only be partially evaluated because of the overlap of the various structures in the craniofacial skeleton, the complexity of which demands considerable expertise in evaluation (Figs. 2.1–2.6).

2.2 Computed Tomography

CT is an X-ray imaging method where the X-ray source rotates around the patient, giving information about the density of the tissues (attenuation profiles) in the slice within the X-ray beam. The attenuation profiles of the slice are Fourier transformed into a matrix of digital values representing a digital image of the slice. Every pixel of the image represents a small volume element (voxel) in the patient. There is density averaging within the voxels (partial volume effects), but no superimposition of structures. The thinner the slice, the lesser are partial volume effects and density averaging. CT permits the analysis of the anatomical structures within the patient without superimposition of structures, and with a relatively good tissue density characterization, which can even be improved by the injection of intravenous contrast material (CM).

¹Contributed by Thomas Treumann, Kantonsspital Luzern (CH), Central Institute of Radiology, Luzern, Switzerland.

Table 2.1 Conventional X-ray techniques for the skull

X-ray	Indication
Skull X-ray in two planes	Cranial fractures
Skull occipito-frontal and occipito-mental	Fractures of the facial skeleton
Occipital exposure (Towne view)	Fractures of the occipital bone
Mandible (Clementsitsch view)	Fractures of the mandible
Mandible unilateral in oblique position	Fractures of the horizontal branch of the mandible
	Tilted collum or fracture of the mandibular condyle
Panoramic X-ray	Collum-condyle-fractures, mandibular fractures, dento-alveolar traumas
Pan-handle X-ray (axial X-ray of the skull)	Fractures of the zygomatic arch
Unilateral exposure of zygomatic bone	
Lateral view of nasal bone	Fracture of the nasal bone

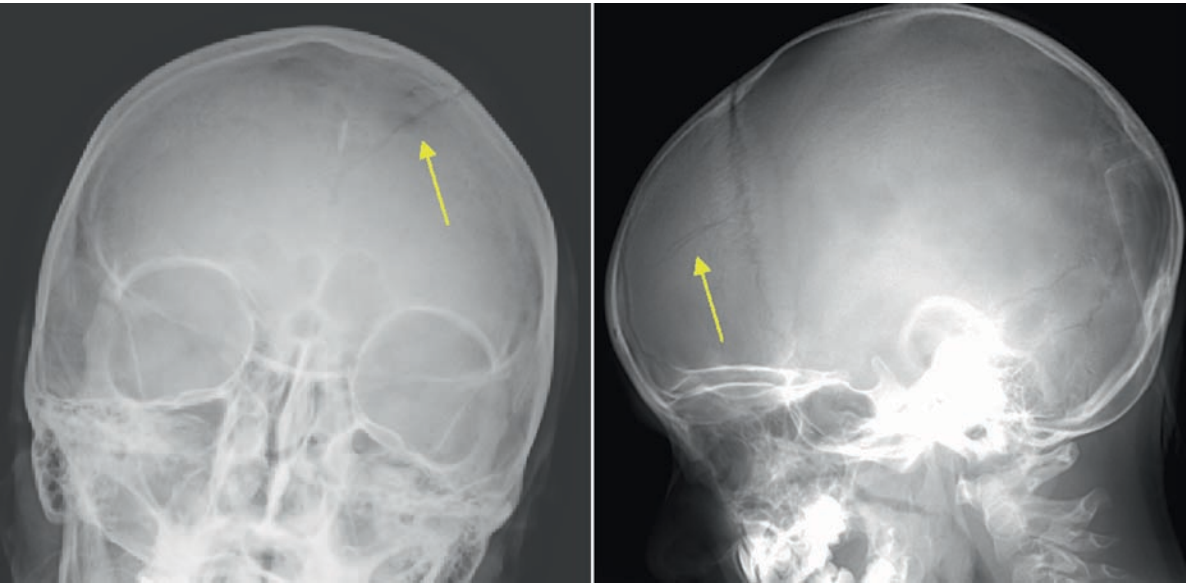


Fig. 2.1 Skull fracture on standard X-ray radiographs. Sharp lucent line without sclerotic margins in left frontal bone, distant to sutures and vascular channels (*arrow*)

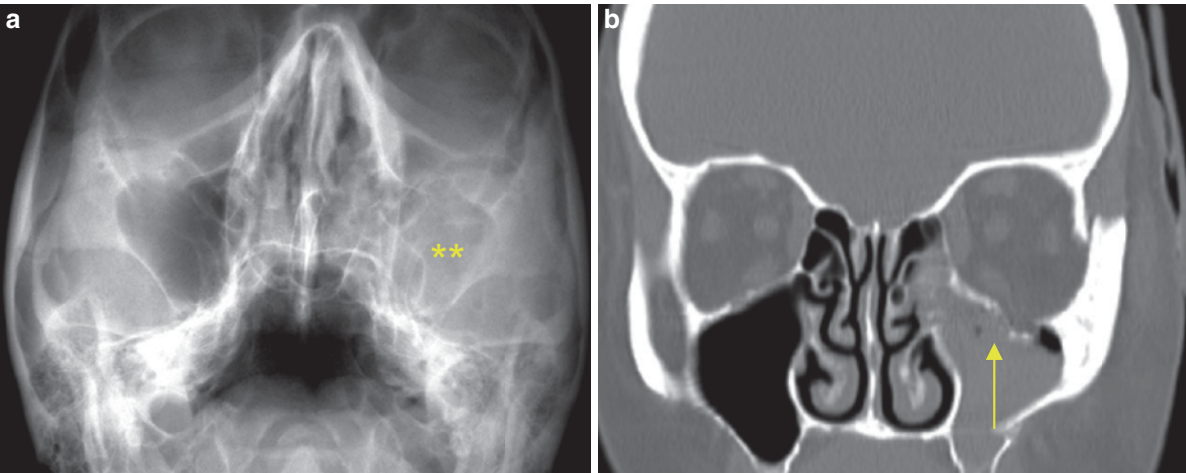


Fig. 2.2 Blow-out fracture of the orbital floor. (a) Indirect fracture sign: total opacification of the right maxillary sinus (*asterisks*). (b) Coronal CT reformatting: depression fracture of the central part of the orbital floor with hemo sinus (*arrow*)

Fig. 2.3 Panoramic X-ray: triple fracture of the mandible. Subcapital collum fracture on the right with dislocation of the capitulum (luxation and massive angulation) (*arrow*) and left neck base fracture without dislocation (*arrow*). Right paramedian corpus fracture (*arrow*)

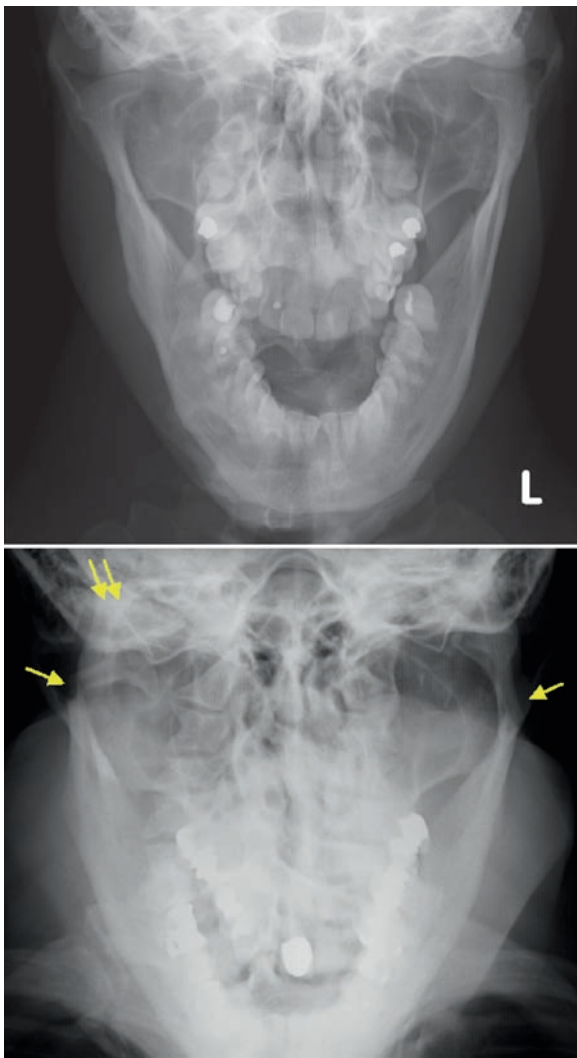
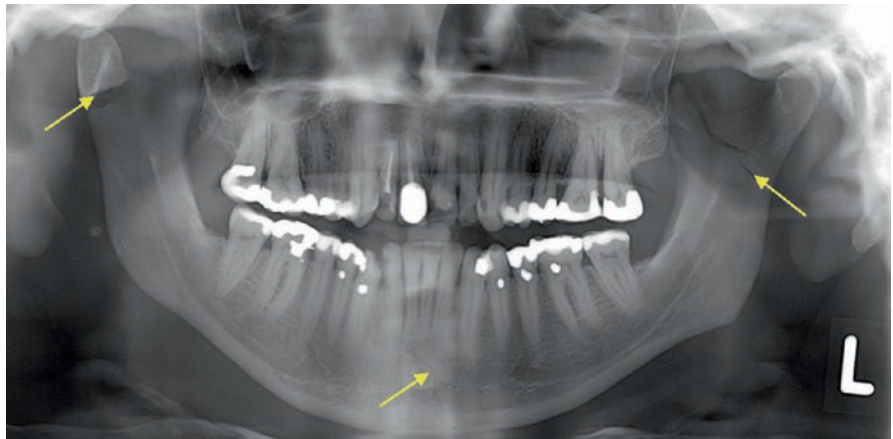


Fig. 2.4 Clementschitsch view of the mandible: *left panel* normal X-ray appearance; *right panel* same patient as in Fig. 2.3. The medial angulation of the right capitulum is well seen in this view (*arrow*). The corpus fracture is superimposed by mediastinal structures and is not seen in this view

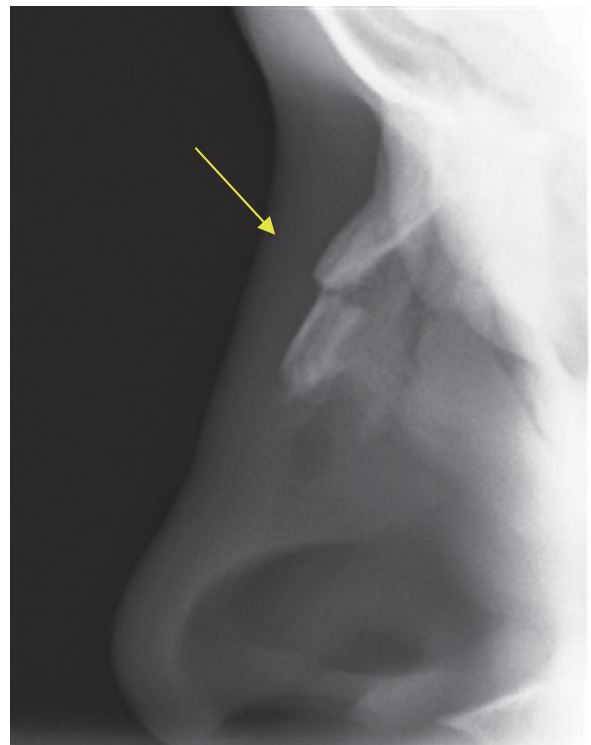


Fig. 2.5 Fracture of the nasal bone with moderate displacement (*arrow*)

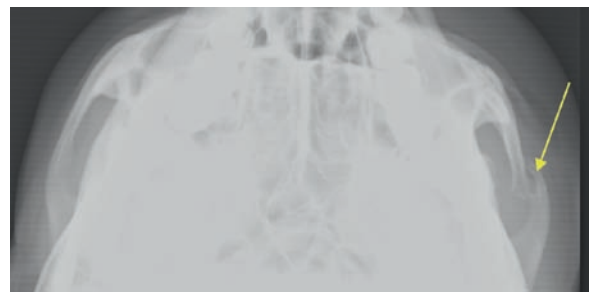


Fig. 2.6 X-ray view of both zygomatic arches. Fracture of the left zygomatic arch (*arrow*)

To cover larger parts of the body, multiple adjacent volumes are acquired. Scanning is done by continuous movement of the patient through the CT gantry in combination with continuous rotation of the X-ray tube, resulting in spiral scanning. This technique is called multislice spiral CT (MSCT). The resulting slices are put together to form a stack, which in turn can be analyzed image by image or by reformatting for interactive analysis in arbitrary imaging planes.

MSCT scanners cover up to 40 mm of patient volume in one rotation, split into up to 128 slices, with slices as thin as 0.5 mm or less. Using MSCT, large

body segments can be scanned within a few seconds with a submillimeter resolution in all three dimensions. The scanners become more powerful from year to year, with an increase in the number of simultaneously acquired slices and in the volume per rotation.

The primary imaging plane of CT images is axial, but many structures are more easily analyzed in other imaging planes. For the evaluation of the facial skeleton, axial and coronal images are mandatory. Until a few years ago, before the MSCT era, the facial skeleton had to be scanned twice, in the axial and coronal direction separately, resulting in a double dose of radiation. In MSCT,

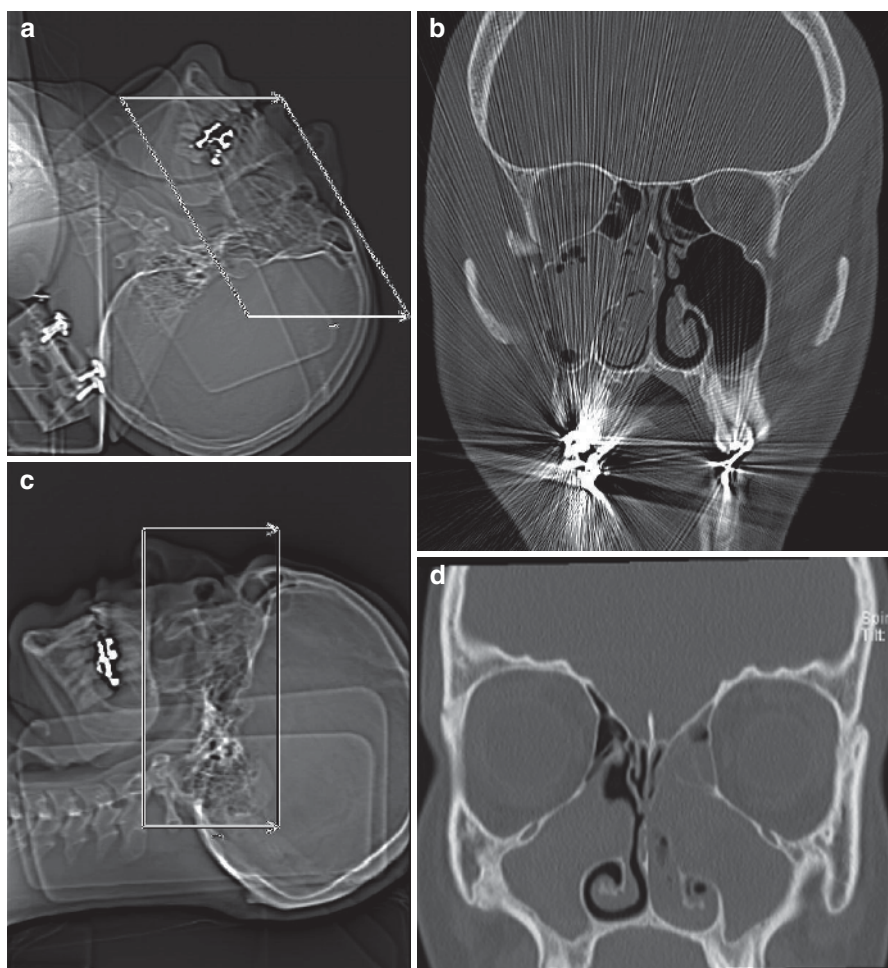


Fig. 2.7 Comparison of direct paracoronal scanning of the midface (a, b) to coronal reformations from thin-slice spiral CT datasets (c, d). Direct paracoronal scanning has been abandoned with introduction of multislice spiral CT scanners. In direct paracoronal scanning, patient positioning is uncomfortable because reclination of the head is required. Furthermore, the CT gantry has to be tilted leaving less space for the patient (a). In the

images, tooth artifacts superimpose relevant structures (b). Axial thin-section CT scanning allows comfortable patient positioning and scanning without gantry tilt (c). Artifacts remain in the plane of the teeth and do not go across relevant structures (d). There is no image quality loss between reformatted images and original paracoronal images (b, d)

only a single dataset in the axial plane is required. The coronal images and any other planes are reconstructed from the axial images by multiplanar reformatting (MPR) on a computer workstation. A workstation can be a CT workstation or a picture archiving and communication system (PACS) workstation. PACS is the electronic image database system with which most hospitals are equipped today. The image quality of the reconstructed coronal images is similar to that of directly acquired coronal images. MPR analysis is routinely used to detect or exclude fractures of the skull base, optic canal, orbital floor, maxilla, palate, and mandible, as well as to measure the extent of dislocations (Fig. 2.7).

In addition to MPR, three-dimensional views of the scanned object can be calculated using shaded surface display (SSD) or volume rendering (VR) algorithms. VR images are color coded and give an impressive view of the anatomy. The three-dimensional (3D) perspectives are valuable for the analysis and visualization of complex fractures. They give an overview over the main fragments and relevant dislocations, from which conclusions about the trauma mechanism can be drawn. On modern computer workstations, 3D views can be calculated within a few seconds, making 3D visualization a practicable routine diagnostic add-on.

In the case of foreign body penetration injuries, CT sensitivity is variable. Whereas glass and metal are seen very well and detected without prior knowledge of their presence, wood and plastic are difficult to detect and special attention must be given for their possible presence. Wood appears like air and plastic materials have different density.

Intraoperatively, CT datasets can be used for navigation. For this purpose, the primary axial CT images are loaded into a computer program which displays the CT findings at the site or during surgery (Hassfeld et al. 1998; Gellrich et al. 1999, 2003). The images have to be loaded in DICOM format from a CD, DVD, or online from the PACS archive, which is the standard format used in medicine (DICOM, digital image communication in medicine). Postoperatively, CT can be used to check and document the repositioned fracture fragments and the position of the osteosynthesis material.

2.3 Magnetic Resonance Imaging (MRI)

MR tomography (MRI, MRT) is an imaging method that uses radiowaves, rather than X-rays, to gain information from the patient. The patient has to be placed in

a high-magnetic-field chamber so as to localize the origin of the radiowaves within the patient's body. Before placing the patient in the chamber, any metal has to be removed from the patient. Cardiac pacemakers and other implanted electronic devices are contraindications for MRI. Anesthetic monitoring requires dedicated equipment with special medical devices, where all metal elements are manufactured out of nonmagnetic materials. These devices are expensive and may not be available in every hospital or MR unit. MR scanning is more time-consuming than CT scanning, and is much less effective in imaging bone than CT.

Because of these limiting characteristics, CT is used rather than MRI when initially examining a trauma patient. However, MRI may be used in evaluating post-operative complications, inasmuch as it lends itself for easy detection of shearing injuries of the brain in the posttraumatic period, which must be suspected when neurologic recovery of the patient is delayed. MRI may be also used to look for cerebrospinal fluid (CSF) leaks, which are a diagnostic problem after skull base injuries. The best, but difficult and invasive, method to localize a CSF leak is to inject contrast medium (CM) intrathecally and to perform CT scans before and after CM administration. MRI is noninvasive, but less sensitive because detection is based on indirect signs, such as fluid in the ethmoid cells or sphenoid sinus. In most cases, a noncontrast-enhanced low-dose CT of the paranasal sinuses and skull base will be sufficient to identify osseous skull base defects, which should be explored by the surgeon.

A rare complication of skull base trauma is a carotid-cavernous sinus fistula. In this case, MRI and MR angiography are helpful in making the diagnosis. Additional treatment is provided by interventional angiography and coil placement (Fig. 2.8).

2.4 Ultrasonography

Ultrasonography is not applicable for adult patients with trauma to the head and face, but may be the method of choice for evaluation in children. Sonographic imaging of the brain in young children is possible through the fontanelles, which are still open. Also, the high spatial resolution of ultrasound allows skull fractures to be detected. As for the facial skeleton, CT is a necessity for treatment decision, thus rendering ultrasound imaging a waste of time (Fig. 2.9).

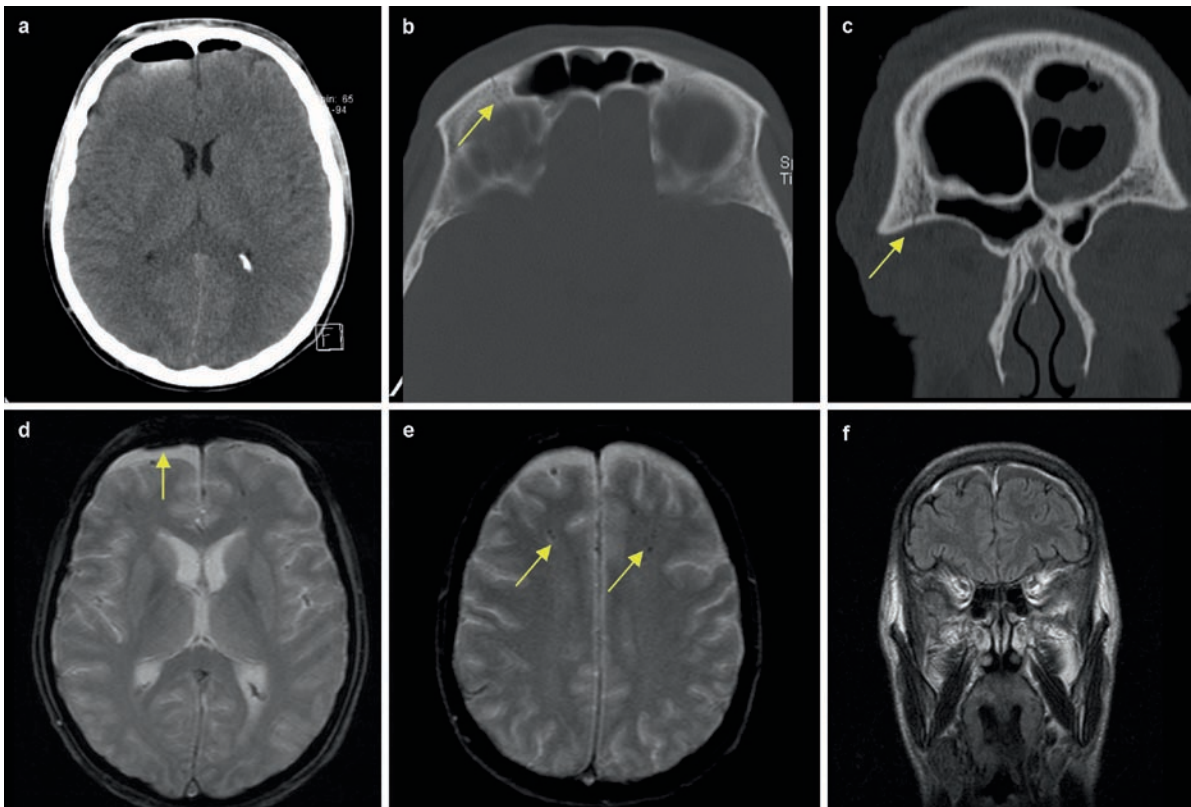
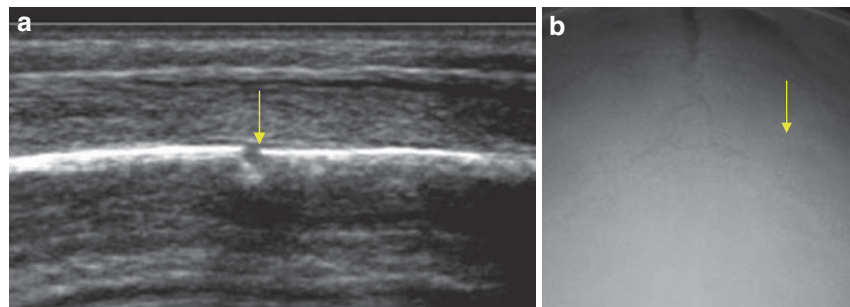


Fig. 2.8 Illustration of the high sensitivity of MRI for shearing injuries and SDH. (a-c) CT after head trauma demonstrating fissural fracture of right orbital roof (arrow) with frontal sinus involvement and pneumatocele. (d, e) MRI demonstrates multiple

small foci of low intensity representing hemorrhage in shearing injuries (arrow). (f) Coronal FLAIR image shows distinctly a small SDH covering both frontal lobes (arrow)

Fig. 2.9 Ultrasonography of a skull fracture. (a) Ultrasound image with cleavage in the tabula externa (arrow). (b) Corresponding X-ray image with evidence of a discrete fracture line on the left parietal bone cranial to the lambdoid suture (arrow)



2.5 Diagnostic Algorithm

2.5.1 General Considerations

Conventional X-ray is no longer the standard in radiological imaging for cranio-facial trauma detection; this is now carried out by CT imaging. CT is widely available and allows fast scanning of the patient. Soft

and hard tissue damage is reliably demonstrated and a first fast overview of the images can be done to identify relevant lesions requiring immediate surgery, such as intracranial hemorrhage or splenic rupture. The CT datasets can then be analyzed thoroughly in an off-line situation at the computer workstation, while the patient is brought to the operating room or otherwise managed by the trauma team. MRI is not the primary imaging modality after trauma, although it is sensitive

for the detection of shearing injuries to the brain, albeit this question is raised later after trauma. Shearing injuries are of little significance in the primary posttraumatic situation (Yokata et al. 1991).

The first important issue to be resolved after cranio-facial trauma is to exclude space-occupying intracranial hemorrhage or increased intracranial pressure (ICP) requiring neurosurgical intervention. This includes evacuation of hematoma, craniectomy, or ICP monitoring. The second point is to assess bone injury (Schneider and Tölly 1984; Bull et al. 1989; Lehmann et al. 2001; Bowley 2003), identifying and classifying fractures.

Technically, the primary CT after trauma is done as noncontrast-enhanced (NECT) scanning. Intravenous contrast administration is contraindicated since it can obscure small intraparenchymal hemorrhages. Contrast-enhanced CT is added only if, based on the NECT scan, an intracranial tumor is suspected or if significant subarachnoid hemorrhage is detected and a cerebral artery aneurysm must be excluded. CM is injected, however, for CT of the cervical spine and trunk; first NECT scanning of the head, followed by scanning other parts of the body. The usual trauma algorithms for CT respect this issue.

The initial CT scan is usually focused on the neurocranium and usually covers the region from the foramen magnum to the apex of the skull. The maxilla is not completely included, and the mandible is usually excluded. However, if significant trauma to the facial skeleton is suspected, the CT technician should be advised to scan the head completely from the chin to the apex. This is not a problem with modern CT systems.

Fractures of the cervical spine must be excluded in any major cranio-facial trauma. The cervical spine can be scanned immediately after the NECT scan of the head without repositioning the patient. It is, however, advisable to apply i.v. CM to exclude dissection of a vertebral artery. In polytraumatized patients, CT is extended to the thorax and the abdomen, also carried out with i.v. CM injection.

2.5.2 Craniocerebral Trauma

2.5.2.1 The Initial CT After Trauma

The primary structure of observation in the initial head CT is the brain. Is there parenchymal bleeding? Are there signs of diffused brain damage? Are there signs

of elevated ICP? Elevated ICP is indicated by narrowing or absence of the external and internal CSF spaces. Narrow spaces may be physiological in young patients. However, absent spaces are never normal, especially if the basal cisterns are not visible. Diffuse brain damage must be suspected if the basal ganglia and cortical structures have the same density as the white matter. This is referred to as “absence of the normal medullocortical differentiation”.

Cerebral hemorrhage usually occurs at the polar areas of the brain and at the brain surface. Typical locations are the frontal and temporal poles, the lateral contours of the temporal lobes, and the basal surfaces of the frontal and temporal lobes. In these regions, the brain collides with the bone or glides over the rough skull base or over the edge of the temporal bone during deceleration.

Hemorrhagic contusions are usually small in the initial CT, but nonetheless always indicate significant brain injury and bear the risk of delayed bleeding, the so-called “blooming-up” of contusional hemorrhages. As a further complication, brain swelling can develop. In order not to miss these complications, CT should be repeated 6–24 h after trauma. The risk of continuing hemorrhage and significant hematomas is high in patients on anticoagulant drugs. In these patients, the CT should be repeated earlier, usually after 4–6 h. The need for ICP monitoring by a surgically placed probe depends on the initial CT findings and is managed by the neurosurgeon. Brain swelling may require immediate or delayed decompression by craniectomy. Also, large hematomas or massive cerebellar swelling in the posterior fossa can result in obstruction of the fourth ventricle and cause hydrocephalus, and may require ventricular drainage (Fig. 2.10).

The second thing to look for is extracerebral hemorrhage. There may be epidural or subdural hematomas (SDHs). Large hematomas with a significant mass effect require immediate surgery. Subarachnoid hemorrhages (SAH) may be present, but almost never require intervention since they generally resolve spontaneously. Still, one should be aware that a SAH may be caused by a ruptured cerebral artery aneurysm, and the rupture of the aneurysm can be the cause for the trauma. If there is significant spread of the SAH in the typical regions around the basal arteries in the basal cisterns, a contrast-enhanced arterial phase CT should be added to look for cerebral aneurysms. If there is no aneurysm on CT, cerebral angiography should be discussed. In the long term,

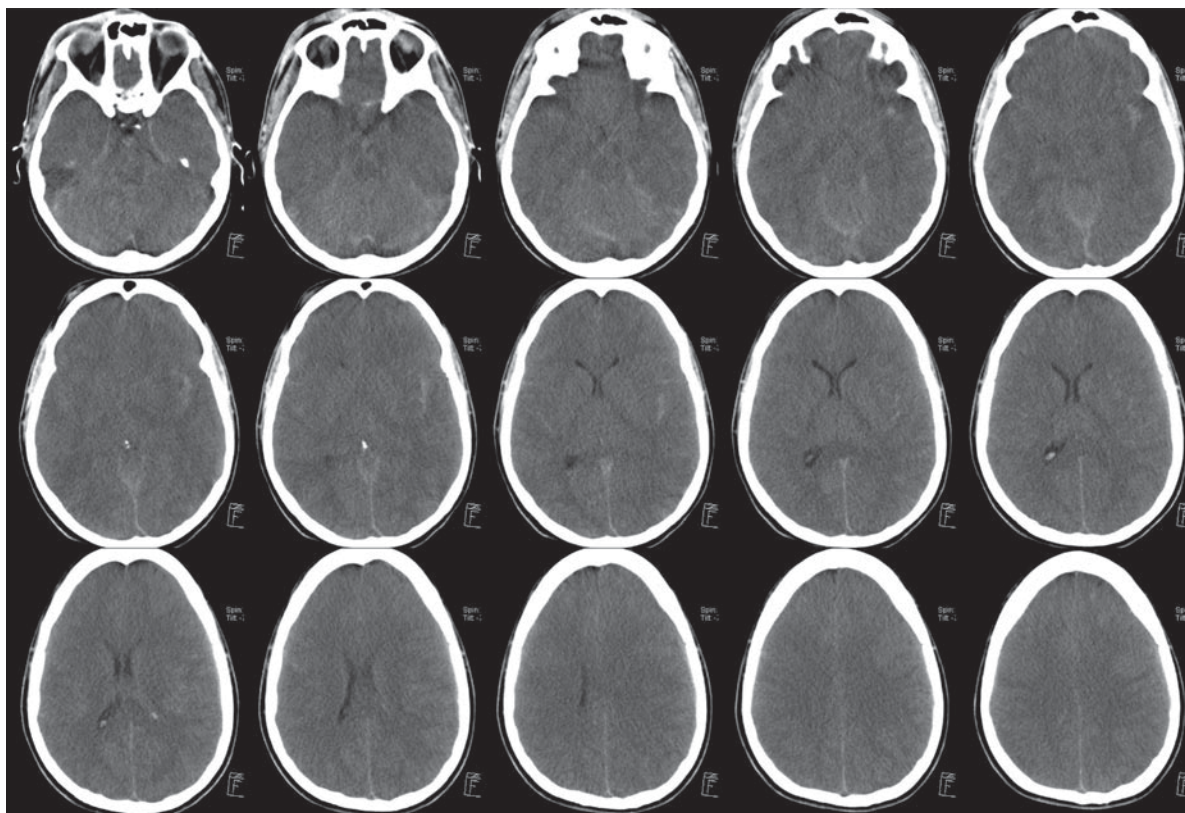
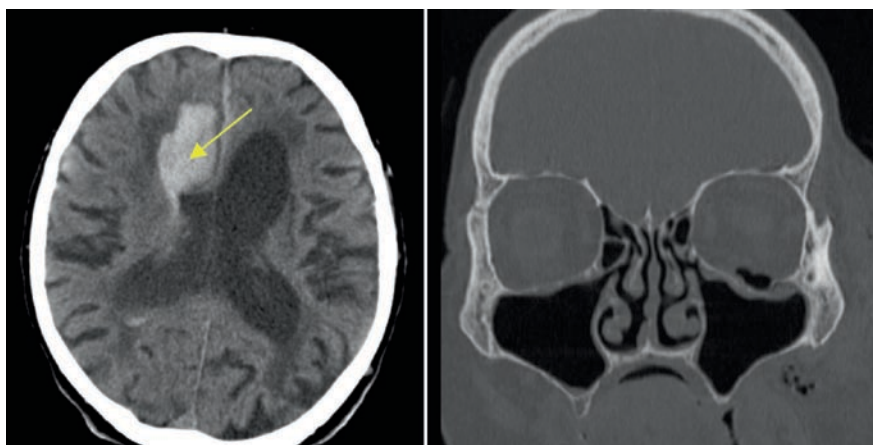


Fig. 2.10 Signs of brain swelling after severe trauma. Compression of the external CSF spaces especially in the tentorial area. Little subarachnoid hemorrhage in the insular cisterne on the left side

Fig. 2.11 Intracerebral hemorrhage (ICH) and midface fracture (left orbital floor); which was first? In this case, the ICH was first and led to collapse of the patient with midface fracture. Location and size of the hemorrhage represent a typical hypertensive bleeding (*arrow*) and not a superficial contusion injury



SAH may cause CSF malresorption and hydrocephalus weeks to months after trauma and require ventricular drainage. Multiple or combined hemorrhages in different areas indicate semi-severe to severe cranio-cerebral trauma. The need for a “second look” CT scan after

12–24 h has already been mentioned. The need for further follow-up CTs will depend on the patient’s clinical course (Figs. 2.11–2.15).

The third thing to look for is fractures. Singular undisplaced skull fractures are of little clinical

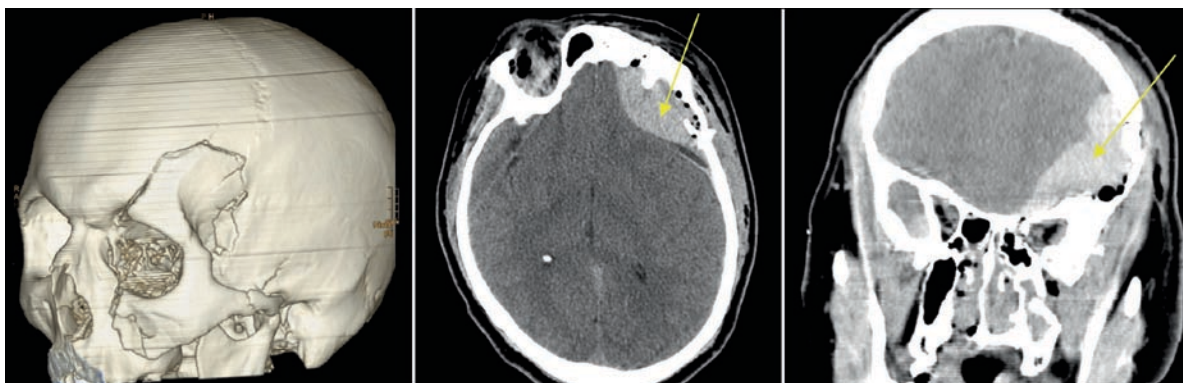


Fig. 2.12 Large supraorbital EDH (arrow) after complex left cranio-orbito-zygomatic fracture

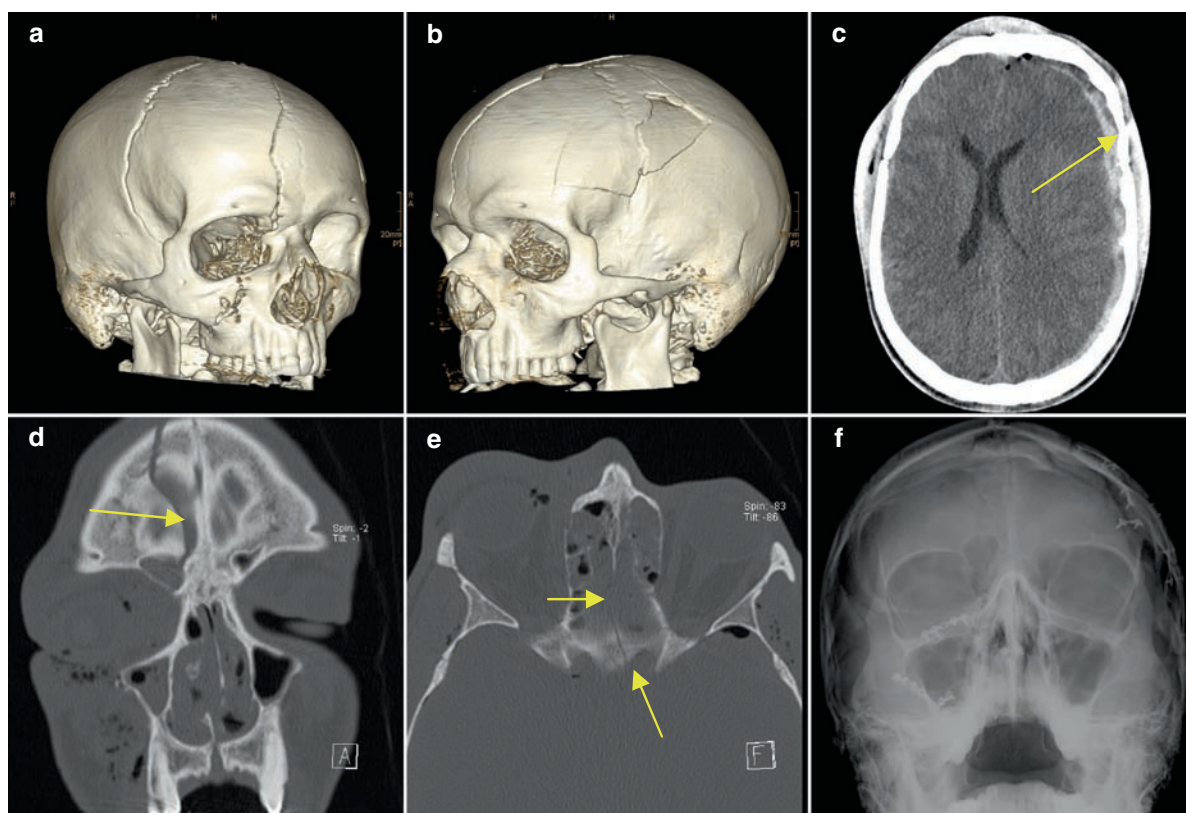


Fig. 2.13 Major burst fracture of the skull after compression injury. Large right-anterior craniofacial skull fragment. (a) Large right-anterior craniofacial skull fragment (b) Left frontoparietal skull impression fracture. (c.) Left subdural hematoma (SDH) (arrow) with midline shift to the right (d, e) The fracture

of the frontal bone continues through the planum sphenoidale, right ethmoid and orbit (arrow) into the anterior wall of the right maxillary sinus (f) Postoperative result after craniotomy (left) and zygomatico-orbital osteosynthesis (right)

significance unless they cause epidural hematomas (EDH). Depressed and displaced fractures with gaps and steps between fragments may require surgery.

In describing a fracture, the first step is to define the affected bone structures:

- Calvarial bones (frontal, temporal, parietal, occipital)
- Anterior and/or posterior wall of the frontal sinus
- Ethmoid (roof, lateral wall)
- Sphenoid sinus, sphenoid wing, optic canal and clivus

Fig. 2.14 Complex bilateral midface fracture and cranio-frontal fracture with little displacement (*arrow*), but massive brain injury. Frontobasal and right temporo-polar contusion hemorrhages (*arrow*). Intraventricular hemorrhage with hydrocephalus (*arrow*). CSF circulation is blocked by the clot in the fourth ventricle leading to slight widening of the temporal horns of the ventricles

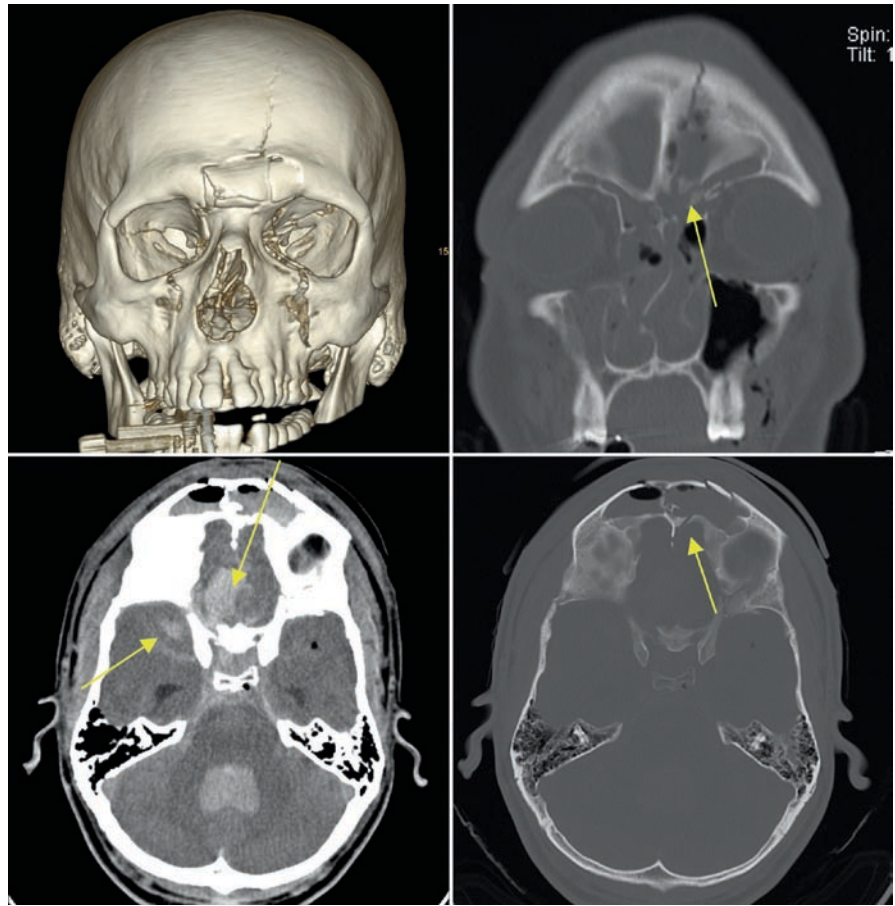


Fig. 2.15 Typical hemorrhagic contusions in both frontal lobes (*arrow*) after midface trauma. Fracture of the left zygomatic arch and lateral zygomatico-maxillary complex

- Orbit (roof, medial wall, lateral pillar, orbital floor, optic canal)
- Nasal bone
- Zygoma and zygomatic arch
- Maxillary sinus (anterior and lateral walls, orbital floor)
- Maxilla (alveolar process, teeth, pterygoid process) and palate

Table 2.2 Radiological findings in trauma CT

<i>Epidural hematoma</i>	Compressed tentorial and basal cisterns
Lens shaped between dura and tabula interna	Compressed fourth ventricle (if hemorrhage is in the posterior fossa)
Usually stops at skull sutures	Hydrocephalus (when the fourth ventricle is compressed)
Requires surgery dependent on size	
<i>Subdural hematoma</i>	<i>Brain swelling</i>
Crescent-shaped	Compressed external CSF spaces over the swollen brain parenchymal area
Along the cranial vault	Narrow ipsilateral ventricle
Along the falx	Mid-line displacement
Along the tentorium	Asymmetry of the tentorial cisterns
Exceeds the skull sutures	
Requires surgery dependent on size	<i>Signs of increased ICP</i>
<i>Traumatic SAH</i>	Compression of external CSF spaces
Blood in the external CSF spaces (sulci or basal cisterns)	Narrowed ventricles
Traumatic SAH is common in severe cranio-cerebral injuries	Compression of the tentorial and basal cisterns: Ambiens cistern (lateral to the midbrain) and quadrigeminal cistern (dorsal to the quadrigeminal lamina)
Clinical significance is low	Foramen magnum filled out with brain parenchyma (cerebellar tonsils)
<i>Nontraumatic SAH</i>	<i>Intracranial air (pneumatocele, pneumatocephalus)</i>
In each SAH: should think about the possibility of a ruptured cerebral artery aneurysm. A rupture may be the cause for the trauma. Check the trauma history	Open brain injury
If there is a suspicion of an aneurysm, perform an Angio-CT and discuss cerebral angiography	Indicates dural laceration
<i>Parenchymal hemorrhage (contusional hemorrhage)</i>	Indicates fracture of temporal bone at the skull base
Common in mid-severe and severe cerebral trauma	Look for:
At surface and on the poles of the brain	Frontal skull base fracture
May “bloom up”	Sphenoid sinus fracture
Require additional CT scan (within next 24 h)	Mastoid fracture
May be accompanied by brain swelling and require decompression surgery	Temporal bone fracture
<i>Signs of space occupying hemorrhage</i>	<i>Foreign bodies</i>
Compressed external CSF spaces on the side of the hemorrhage	Following penetration injuries
Compressed lateral ventricle on the hemorrhage side	Glass: Most often superficial in skin
Displacement of the midline to the contralateral side	Wood: Difficult to detect, because of appearance like air/emphysema
	Metal: May cause artifacts

- Mandible
- Temporal bone and mastoid

The second step is to define dislocations: impressions, overlaps, and malalignments of the relevant structures. In the CT analysis, one should check the following (Table 2.2, Fig. 2.16):

- Skull contours
- Nasion
- Supraorbital margin
- Infraorbital margin
- Lateral orbital wall
- Zygoma
- Zygomatic arch
- Anterior nasal spine

2.5.3 Skull Base Fractures

There is a high coincidence of midface fractures and skull base fractures. The skull base is mostly affected in the frontobasal and fronto-ethmoidal regions.

- The high coincidence of facial skeletal fractures and frontobasal and fronto-ethmoidal injuries in midfacial traumas requires a CT scan to evaluate the skull base (Joss et al. 2001; Bowley 2003).

Fracture of the skull base can be the direct extension of skull fractures or orbital fractures into the skull base. For example, a temporal bone fracture can extend into the temporal skull base; a frontal bone fracture can radiate into the orbital roof, ethmoid and sphenoid; or

Craniocerebral trauma

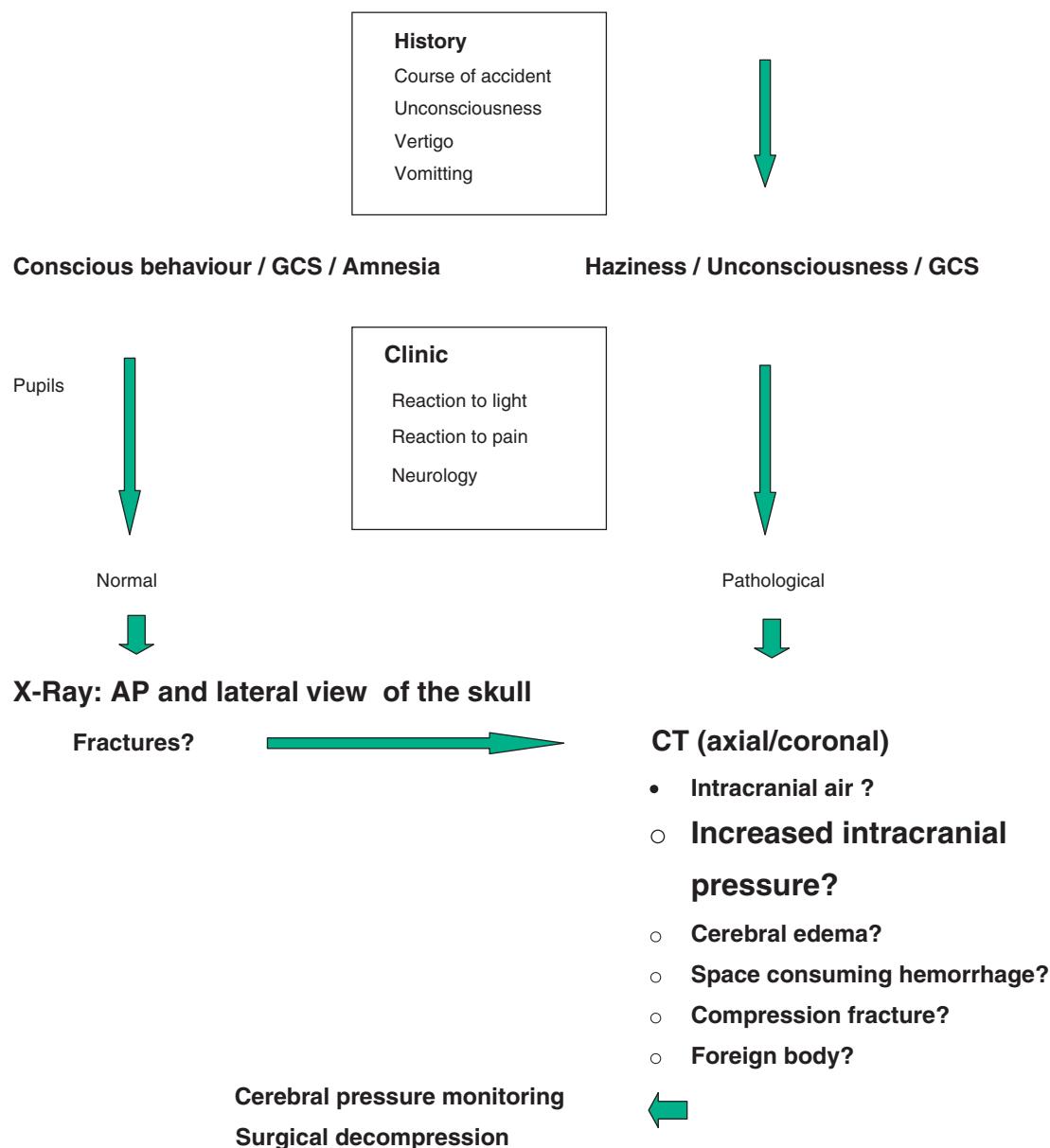


Fig. 2.16 Radiological – diagnostic procedure in craniocerebral trauma – flow chart

an occipital fracture can radiate down into the foramen magnum. Anterior head trauma can result in complex fractures of the frontal skull base and ethmoid bone and may extend into the roof of the sphenoid sinus, the clivus and the sella. Temporal bone fractures can radiate into the petrous bone and mastoid process and

cause hemorrhage in the mastoid cells and tympanon. Clinical symptoms are otic hemorrhage, otic liquor-rhea and hearing loss.

Another mechanism leading to skull base fractures is the indirect energy transmission from the mid-face to the skull base through the main vertical pillars. This

Skull base fractures

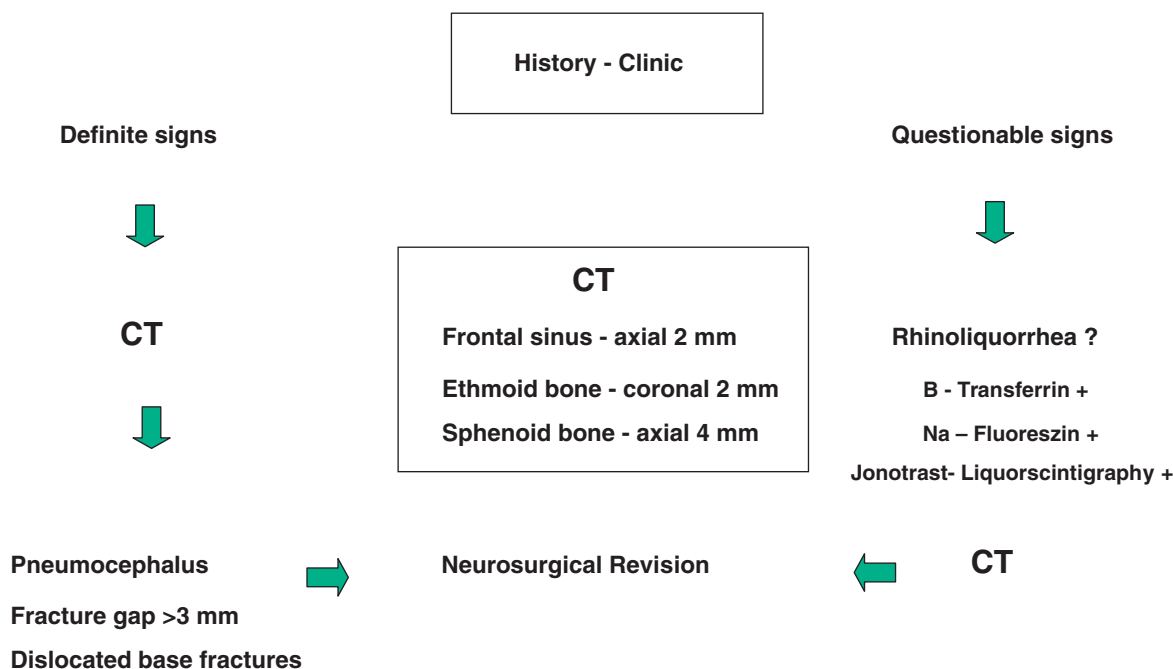


Fig. 2.17 Radiological – diagnostic procedure in skull base fractures – flow chart

mainly affects the temporal skull base and the ethmoid. Not associated with mid-face fractures are skull base fractures after axial head trauma from the vertex with fractures in the region of the foramen magnum and the risk of a burst fracture of the first cervical vertebra (atlas ring burst fracture).

There are direct and indirect signs of skull base fractures. Direct signs are fracture lines, fracture gaps and steps between fragments. Indirect signs are intracranial air collections and liquorrhea. Intracranial air collections can be demonstrated in 25–30% of skull base fractures (Probst and Tomaschett 1990). Small air collections are regularly seen with fractures of the temporal bone and sphenoid sinus. Vast air collections (pneumocephalus) occur after destructive fractures of the frontal sinus and ethmoid roof. In the CT dataset, the primary axial images are most helpful to detect skull base fractures and must be analyzed thoroughly. To exclude undisplaced skull base fractures, MPR is required. MPR is also required for analysis of the extent of displacement of skull base fractures. Coronal images should be routinely reconstructed from the axial image set by the CT technician's team (Fig. 2.17).

2.5.4 Midface Fractures

For midface fractures, CT images in the axial and coronal planes are obligatory to differentiate fracture types and to define the extent of the fracture. The sagittal plane may be helpful to assess dislocations in the anterior-posterior direction (nasion, maxilla). Oblique sagittal images parallel to the optic nerve or parallel to the inferior rectus muscle of the orbit may be helpful to visualize muscle entrapment in fractures of the orbital floor. The required series of images should be generated by the CT technician. In addition, analysis can be done interactively in a PACS viewer, if available.

CT permits a differentiated fracture assessment and provides evidence of injury in anatomically difficult areas, e.g., the orbits, the naso-orbito-ethmoidal complex, the peri- and retroorbital skull base and the retro-maxillary region (Terrier et al. 1984; Schwenzer and Pfeifer 1987; Schneider and Tölly 1984; Manson et al. 1990; Whitaker et al. 1998; Rother 2000).

Classification of midface fractures, according to the classification systems outlined in Chap. 3, surgical planning and intraoperative navigation are based on CT.

Midface fractures

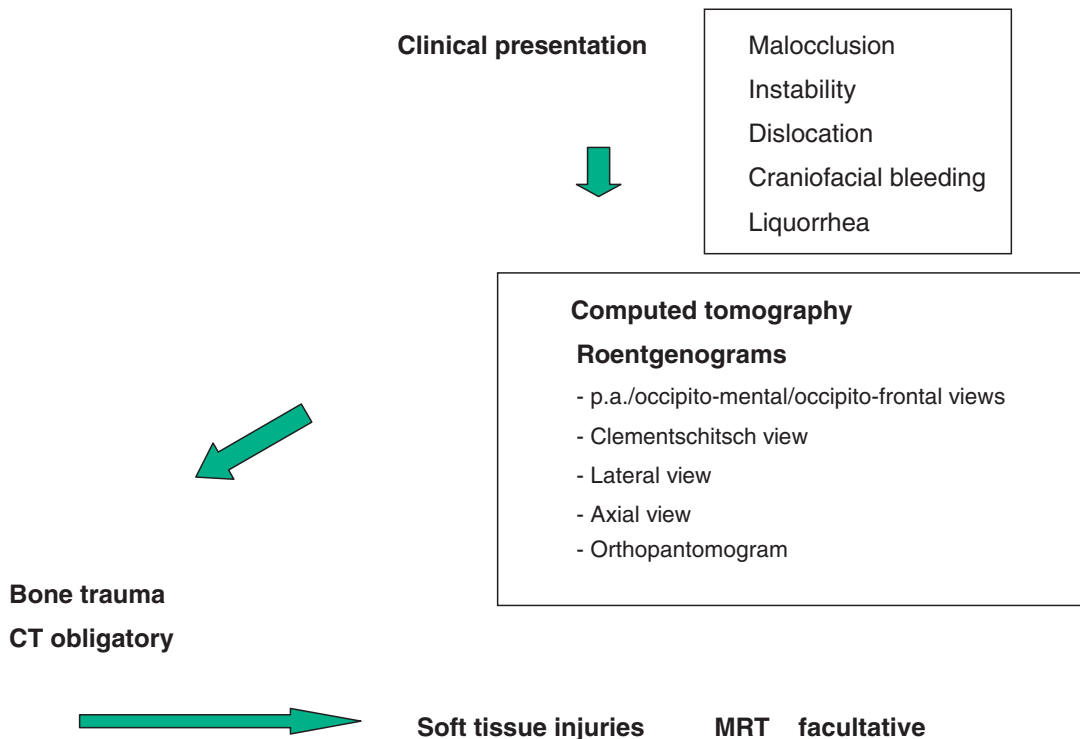


Fig. 2.18 Radiological – diagnostic procedure in midface fractures – flow chart

Axial images should be scrutinized for:

- Fractures of the anterior and posterior walls of the frontal sinus
- Fracture of the lateral orbital wall
- Fracture of the medial orbital wall (blow-out fracture)
- Ocular lens luxation or rupture of the ocular bulb
- Fracture and dislocation of the nasal bone
- Fractures of the maxillary sinus with hematosinus
- Hematosinus without apparent wall fracture may indicate fracture of the orbital floor
- Fractures of the anterior lateral walls of the maxillary sinus are associated with inward rotational dislocation of the zygoma
- Fracture of the zygomatic arch
- Fracture of the alveolar crest of the maxilla and of the palate bone
- Mandibular fractures (ramus)

Particular to detection in the coronal images are:

- Fractures of the orbital floor
- Fractures of the orbital and ethmoid roofs (frontal skull base)

- Fracture of the hard palate
 - Fracture of the pterygoid process
 - Mandibular collum or condyle fractures
- Sagittal CT-scan display (Fig. 2.18):
- Depressed fractures of the anterior and posterior frontal sinus walls
 - Displacement of the nasal bone into the ethmoid
 - Depressed fracture of the maxilla
 - Sella fractures (rare)

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Craniofacial traumatology comprises:

- Combined fractures of the visceral- and neurocranium
- Posttraumatic craniofacial defects and deformations

Fractures of the craniofacial skeleton comprise variable fractures of the anterior skull base, the frontoglabellar region, the orbit and the maxillary complex (Weerda 1995) (Fig. 3.1).

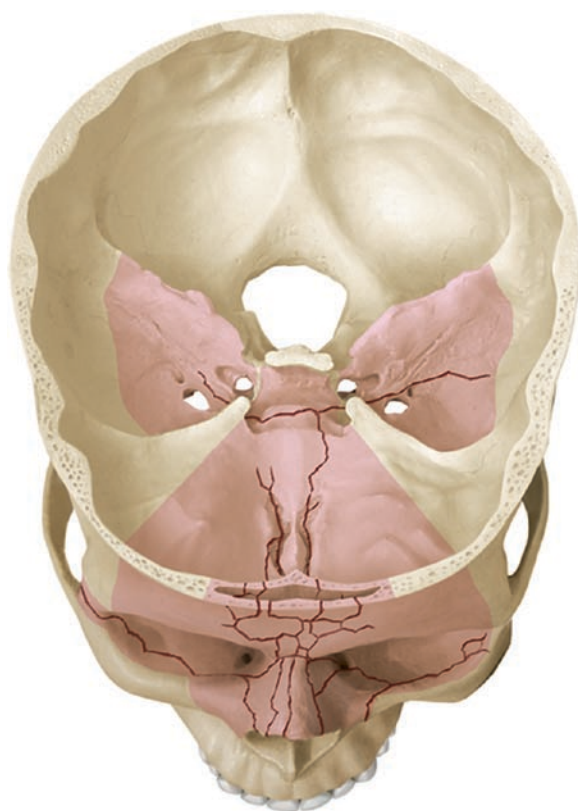


Fig. 3.1 Fractures of the craniofacial skeleton consist of variable fractures of the anterior skull base, the frontoglabellar region, the orbit and the maxillary complex (mod.a. Weerda 1995)

Fractures affecting the skull base are the most common form of neurocranial injuries, particularly in midface fractures type Le Fort II and III, respectively Wassmund III and IV (Hausamen and Schmidseider 1975; Machtens 1987; Manson 1986, 1998; Joss et al. 2001).

The neurocranial involvement ranges from facultative skull base fractures in subcranial midface fractures to extensive craniofacial skull fractures with obligatory frontobasal and frontofacial poly-fragmentation in combination with dura- and intracranial soft tissue injuries (Manson 1986; Weerda 1995).

The following *trauma compartments* (midface fracture levels according to Le Fort and Wassmund) with *frontobasal involvement* are differentiated:

- Subcranial midface fractures – frontobasal fractures
Fracture levels: II/III/I + II + III/II + III
- Craniofacial fractures – frontobasal fractures
Fracture levels: II + VI/III + IV/II + III + IV
- Frontofacial fractures – frontobasal fractures
Fracture level: IV (Fig. 3.2)

3.1 Frontobasal: Frontofacial Fractures

Fractures of the frontofacial level (fracture level IV) include skull base fractures in the region of the anterior cranial fossa with involvement of the neighboring aerated sinuses and fractures of the frontal and glabella structures, frequently including the orbital roof.

These fractures most often occur in the frontal region, including the anterior and/or posterior wall of the frontal sinus. As combined fractures, frontofacial fractures involve the anterior and middle third of the ethmoid (naso-ethmoidal fracture) or the midface (Raveh et al. 1988).

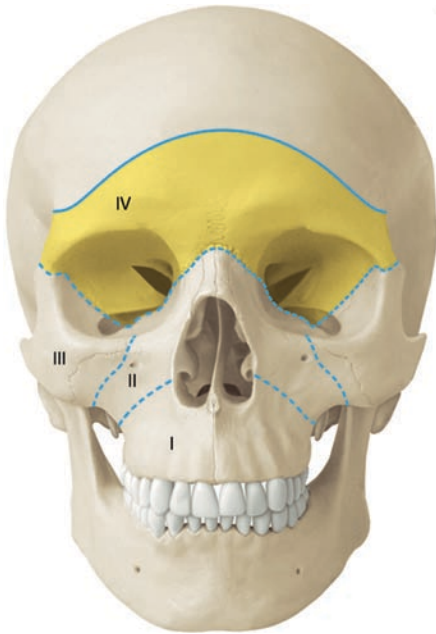


Fig. 3.2 Subdivisions of the midfacial skeleton into three compartments: central (I–II), lateral (III) and frontofacial (IV) (mod. a. Hardt et al 1990)

- Specific pathogenetic danger exists in frontobasal and cranio-cerebral injuries when the subbasal paranasal sinuses are involved in the course of the fracture, providing an interconnection between the intracranial space and the paranasal sinuses, so increasing the risk of an ascending infection.

3.1.1 Anatomical Classification: Skull Base Fractures

Escher introduced a classification for frontobasal fractures according to the fracture site, the extent of the fracture and according to the direction of the traumatic force acting. He defined four different types of frontobasal fractures: high, middle, low, and lateral fractures — types 1–4, respectively (Escher 1969, 1971, 1973).

3.1.1.1 Type 1

Extensive high frontobasal fracture resulting from a direct craniofrontal impact to the upper frontal bone radiating into the paranasal sinuses:

Extended frontobasal comminuted fractures with disintegration of the frontal bone and participation of

the posterior wall of the frontal sinus as well as the ethmoid roof combined with dura lacerations and brain injuries (Probst 1971; Boeninghaus 1974; Schwab 1995; Weerda 1995).

3.1.1.2 Type 2

Localized mid-frontobasal fracture resulting from circumscribed violence applied to the fronto-naso-basal region:

Localized depressed fracture or bony comminution in the frontal sinus/ethmoid region primarily in the region of the cribriform plate, the crista galli, the posterior ethmoid and the roof of the sphenoid with dural injuries (Probst 1971; Boeninghaus 1974; Schwab 1995; Weerda 1995).

3.1.1.3 Type 3

Low frontobasal fracture with subcranial midface avulsion resulting from violent force acting on the midface:

Midface avulsion from the skull base in the context of midface fractures, particularly in Le Fort II, Le Fort III, Wassmund IV and Wassmund III levels, whereupon depending on the applied force, extensive particles of the frontal base may be shattered and dislocated (Probst 1971; Boeninghaus 1974; Schwab 1995; Weerda 1995; Ernst et al. 2004).

3.1.1.4 Type 4

Latero-orbital frontobasal fracture from forces acting on the orbito-temporal region with fracture of the frontal sinus and dislocation of latero-frontal parts of the orbital roof (Probst 1971; Boeninghaus 1974; Weerda 1995; Schwab 1995).

3.1.2 Topographic Classification: Skull Base Fractures

For therapeutic purposes, the modern classification of frontobasal fractures relates to the frontobasal topographic diagnosis, which correspondingly classifies the individual regions of the paranasal sinuses as follows (Oberascher 1993; Schroeder 1993):

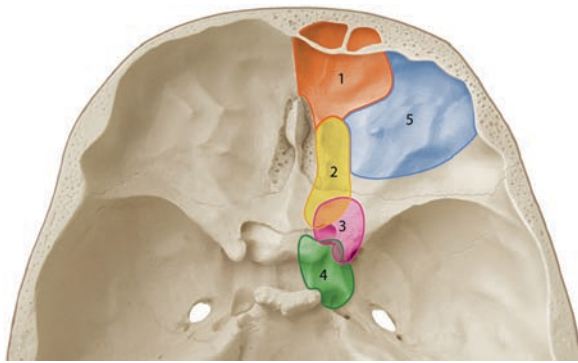


Fig. 3.3 Classification of frontobasal fractures based on anatomical regions (1–5) (mod. a. Oberascher 1993)

- Region 1: Posterior wall of the frontal sinus
- Region 2: Anterior ethmoid–cribriform plate
- Region 3: Posterior ethmoid roof
- Region 4: Roof and lateral wall of sphenoid sinus and adjacent petrous part of the temporal bone
- Region 5: Orbital roof

This classification is suggestive for the surgical approach, as it is not categorized according to fracture type but according to the topographical unilateral or bilaterally affected areas of the anterior skull base and so determines which region needs attention (Fig. 3.3).

- The classifications mentioned refer mainly to the course of frontobasal fractures; however, they do not include extensive cranio-maxillary fractures or serious panfacial fractures (PFs), which may not only present a midface avulsion from the skull base but also accompanying demolition of the upper, middle, and lower facial levels as well as the neurocranium with extensive basal and cerebral injuries.

3.2 Midface Fractures

Midface fractures may be classified according to parameters as follows:

- Fracture level
- Topographic region and
- Extension of the fracture

Degree and direction of the applied traumatic force, as well as impact angle and the area struck, all influence the resulting injury to the midface and hence lead to varying types of fractures, varying in extent (Schwenzer and Pfeifer 1991).

3.2.1 Standard Classifications

Various classifications exist in midface fractures:

1. The widely used classification according to Le Fort (Le Fort 1901) is based on a combination of characteristic, mostly transverse fracture lines, which are defined by the traumatic impact on the center of the face in relation to anatomical structures on different levels of height:
 - Le Fort I fractures (low-level fracture)
 - Le Fort II fractures (pyramidal fracture)
 - Le Fort III fractures (high-level fracture) (Fig. 3.4)
2. The *topographic classification* in *central, centrolateral, and lateral* midfacial fractures is internationally recognized (Mathog 1984; Rowe and Williams 1985; Bowerman 1985; Lew and Sinn 1991; Fonseca and Walker 1991; Prein et al. 1998; Schwenzer and Ehrenfeld 2002). The following fracture types are subsumed in these classifications:
 - Central midface fractures
Le Fort I, II, sagittal midface fractures, Wassmund I, II, III fractures, naso-ethmoidal fractures, naso-maxillary fractures
 - Centrolateral midface fractures
Le Fort III, Wassmund IV fractures
 - Lateral midface fractures
Zygoma, zygomatic arch fractures, zygomatic-orbital fractures
 - Combined midface fractures

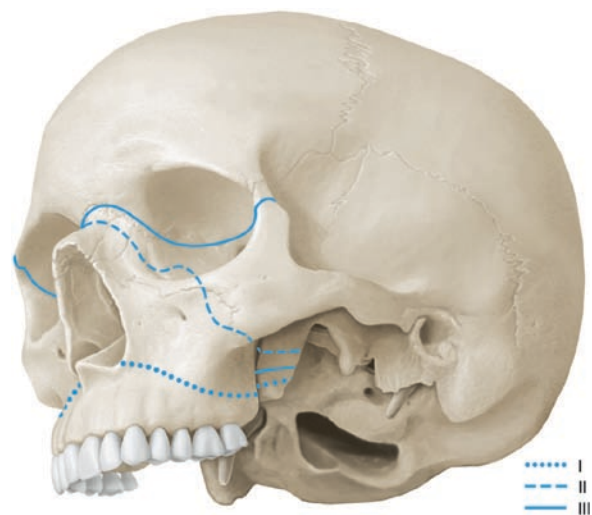


Fig. 3.4 Fracture levels of Le Fort I–III transverse subcranial midface fractures (mod. a. Schwab 1995)

Orbito-maxillary fractures, maxillo-mandibular fractures, zygomatic-mandibular fractures, zygomatic-orbital-maxillary fractures, zygomatic-orbital-cranial fractures, fronto-maxillary fractures, PFs.

3.2.2 Central Midface Fractures

Central midface fractures include fractures of the Le Fort I- and II-type. The central midface block is unhinged from the rest of the facial skeleton and dislocated posteriorly or maybe even wedged. The dislocation is partially determined by the direction of the applied force and partially by the muscular tension applied by the pterygoid muscles. Both pterygoid muscles pull the mobile midface section dorsally and caudally, so resulting in an occlusal disturbance.

With an additional sagittal fracture of the central midface complex, a diastema forms between the incisors and more severe dislocations can even result in a traumatic cleft palate (Lew and Sinn 1991; Fonseca

and Walker 1991; Ewers et al. 1995; Prein et al. 1998; Schwenzer and Ehrenfeld 2002).

Fracture course: Le Fort I/Wassmund I fracture

The line of fracture runs from the anterior bony aperture of the nose above the nasal spine through the facial wall of the maxillary sinus, the zygomatico-alveolar crest, the maxillary tuberosity, through the caudal apex of the pterygoid process, through the medial nasal wall of the maxillary sinus and then reaches the point of exit again at the anterior bony aperture of the nose.

As a general rule, the vomer is affected and the cartilaginous nasal septum is often dislocated. Occasionally the pterygoid does not fracture, instead the maxilla avulses at the pterygo-maxillary junction (Fig. 3.5).

In Le Fort II/Wassmund II and III fractures, the maxilla, nasal skeleton, ethmoid, and lacrimal bones are quarried out of the midface as a pyramid. Subcranial involvement is frequent (Ewers et al. 1998; Prein et al. 1998; Schwenzer and Ehrenfeld 2002).

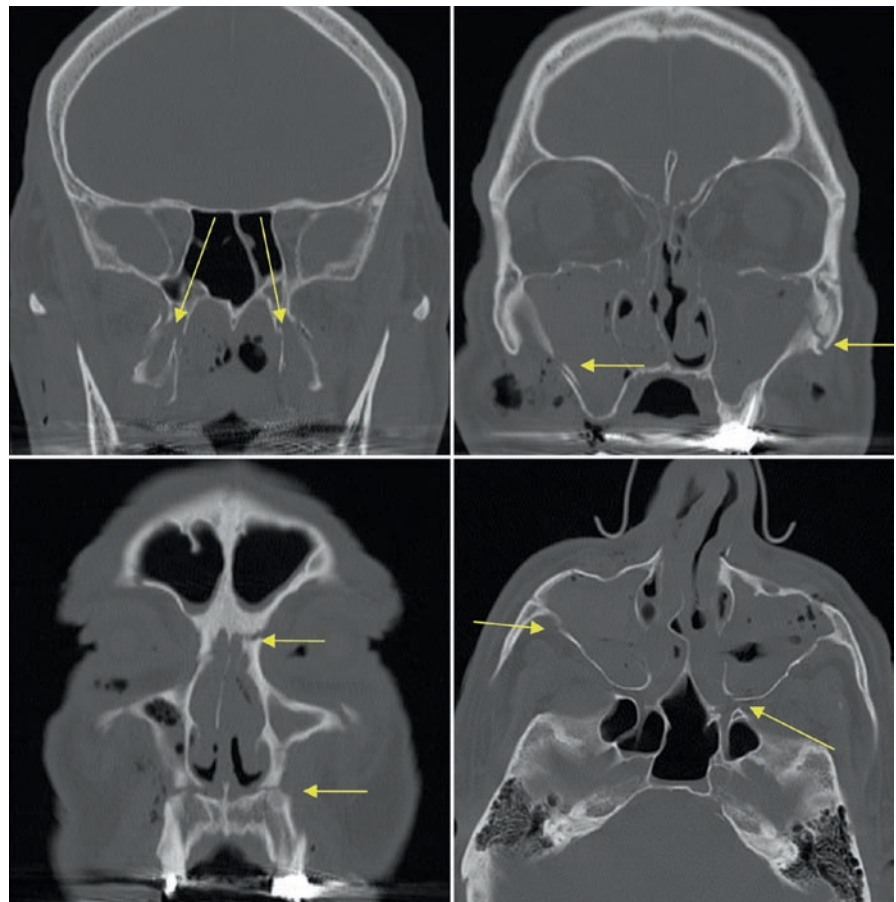
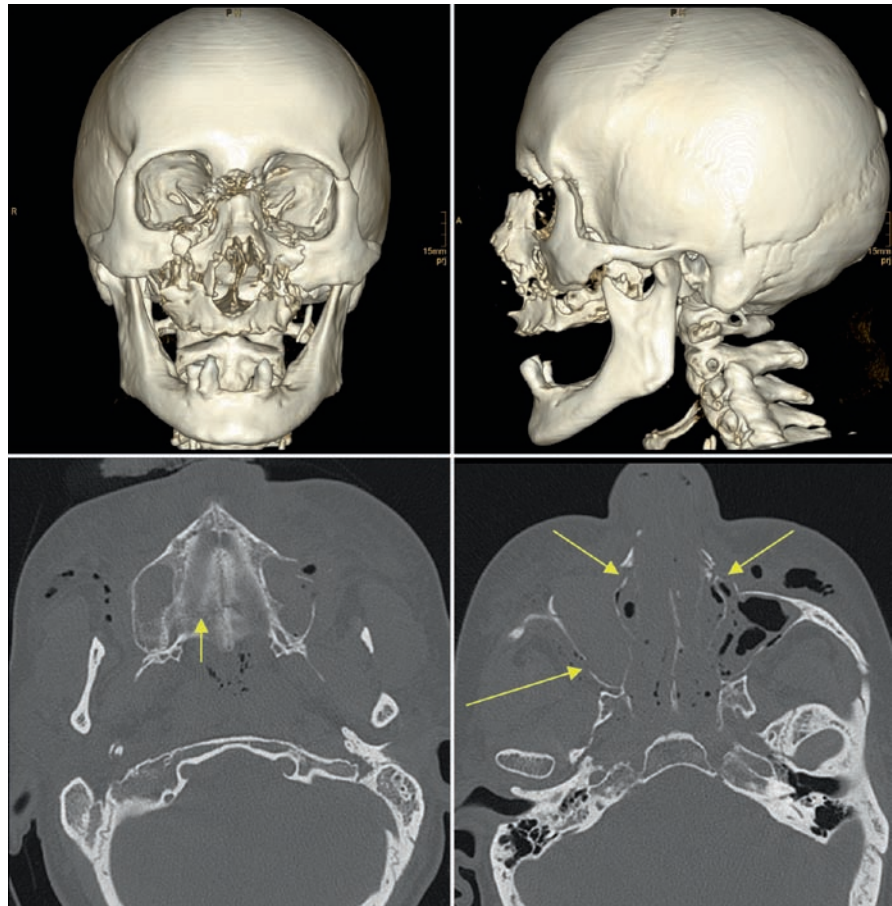


Fig. 3.5 Le Fort I-II fracture: horizontal fractures through the maxillo-frontal pillars, the lateral walls of the maxillary sinuses and the pterygoid processes (arrows) with separation of the maxillary body

Fig. 3.6 Le Fort II fracture: separation of the midface from the frontal base and the zygoma with mobilization of the maxilla, nose, and ethmoid (*arrow*). Transverse fracture of the hard palate (*arrow*)



Fracture Course: Le Fort II/Wassmund II and III Fracture

The line of fracture in Le Fort II/Wassmund II fractures runs above or within the nasal bones, the medial orbital wall, along the inferior orbital fissure, through the infra-orbital canal, infra-orbital foramen and facial wall of the maxillary sinus and usually splits the pterygoid process in the middle third.

In Wassmund III fractures, the nasal bone is not included in the fracture segment. The fracture runs from the orbital floor across the maxillary frontal process to the cranial border of the anterior aperture of the nose. The remaining fracture course is identical to that of the Le Fort II fracture.

Course variations are common in these fractures. Extensive dislocations and mid-facial impressions involving the *ethmoid bone, the orbit and the nasolacrimal ducts, i.e., the naso-orbito-ethmoidal (NOE) complex*, are not uncommon. Concurrently there are often comminuted fractures of the nasal framework or septum luxations as well as fractures of the osseous

components of the nasal septum, the vomer and the perpendicular plate of the ethmoid bone (Fig. 3.6).

3.2.3 Centrolateral Midface Fractures

Centrolateral midface fractures, i.e., subcranial Le Fort III fractures, result in a secession of the viscerocranium from the neurocranium (Ewers et al. 1995; Prein et al. 1998; Schwenzer and Ehrenfeld 2002).

Fracture course: Le Fort III/Wassmund IV fracture

Beginning at the fronto-nasal and fronto-maxillary sutures, by-passing the optical canal, the lacrimal bone and medial orbital wall are disrupted. The line of fracture continues through the inferior orbital fissure and bifurcates, on the one hand, dorso-medially through the pterygopalatine fossa to the base of the pterygoid process, and, on the other hand, antero-laterally to the

lateral orbital margin along the zygomaticosphenoidal suture as far as to the fronto-zygomatic suture. The frontal bone is herewith separated from the zygoma and the maxilla.

In addition, the connection between the zygomatic arch and the temporal bone is disrupted and the osseous nasal septum fractures directly under the skull base. In *Wassmund IV fractures*, the nasal compartment is omitted from the fracture course and the fracture continues along the naso-maxillary suture.

3.2.4 Skull Base and Fracture Levels in the Region of the Septum

Among the *subcranial fractures*, the Le Fort II and III fractures and the Wassmund III and IV fractures differ in distance to the anterior skull base (Becker and Austermann 1981; Joos et al. 2001).

In the *Le Fort II fracture*, the naso-septal fracture line runs closer to the base through the superior

perpendicular plate, vomer, and the cranial pterygoid process; likewise *Wassmund III fractures*, but these run more distant from the skull base at a considerable distance below the skull base through the inferior perpendicular plate and vomer and split the pterygoid process in the middle section.

In *Wassmund IV fractures*, the medio-sagittal fracture of the nasal septum runs slightly lower than in the *Le Fort III fractures*, though close to the base in the osseous septal region.

However, the *Le Fort III fracture* runs immediately below the skull base. Both split the pterygoid process in its cranial portion (Rowe and Williams 1985; Schwenzer and Ehrenfeld 2002) (Fig. 3.7).

3.2.5 Lateral Midface Fractures

Lateral midface fractures dis sever the connection between the zygomatic bone and the frontal bone cranially, the

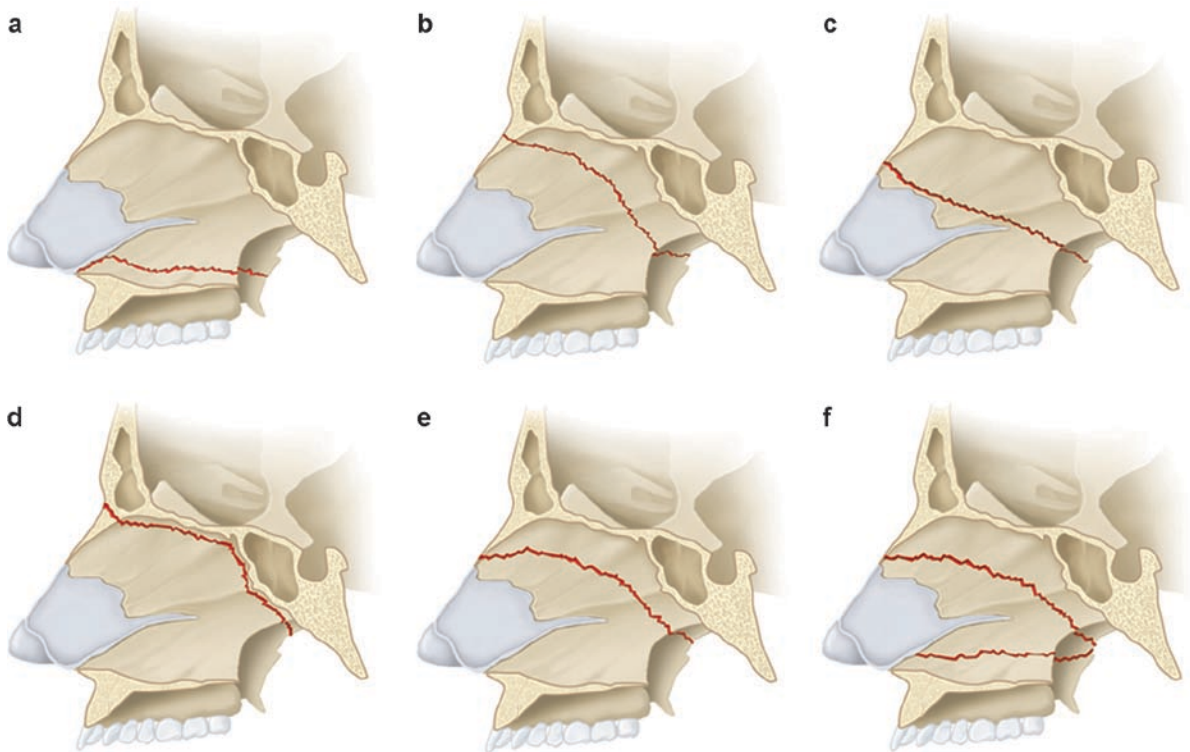


Fig. 3.7 Relation between anterior cranial base and subcranial midface fractures in the region of the nasal septum (mod. a. Schwenzer and Ehrenfeld 2002). (a) Le Fort I, (b) Le Fort II, (c)

Wassmund III, (d) Le Fort III, (e) Wassmund IV, (f) combination: Wassmund IV and Le Fort I

greater wing of the sphenoid bone medially, the maxilla caudally and from the temporal bone dorsally (Lew and Sinn 1991; Fonseca and Walker 1991; Ewers et al. 1995; Prein et al. 1998; Schwenzer and Ehrenfeld 2002).

Fracture Course: Lateral Midface Fractures

In isolated zygomatic fractures, the line of fracture runs through the zygomaticofrontal suture, along the lateral orbital margin, and descends along the lateral and anterior orbital floor, to the infraorbital margin. From here, it proceeds across the facial wall of the maxillary sinus and frequently through the infraorbital foramen to the zygomaticoalveolar crest, then further across the dorso-lateral wall of the maxillary

sinus, back towards to the inferior orbital fissure. In addition, the zygomatic arch fractures. The orbital floor is regularly involved in zygomatic fractures, corresponding to the line of fracture (Figs. 3.8–3.10).

Depending on the applied forces, strong violence may lead to *zygomatiko-orbital comminuted fractures* with polyfragmentation and dislocation of the central zygomatic complex. In the process, the zygomatic body is dislocated into the maxillary sinus, seldomly into the orbit. Collaterally the *facial and dorsolateral wall of the maxillary sinus*, as well as the *orbital floor* and the *medial, more seldomly the lateral orbital walls*, are demolished. Accompanying orbital injuries, further injuries such as *ruptured bulbus* and injuries to the *optical nerve* are possible (Lew and Sinn 1991; Raveh et al. 1992).

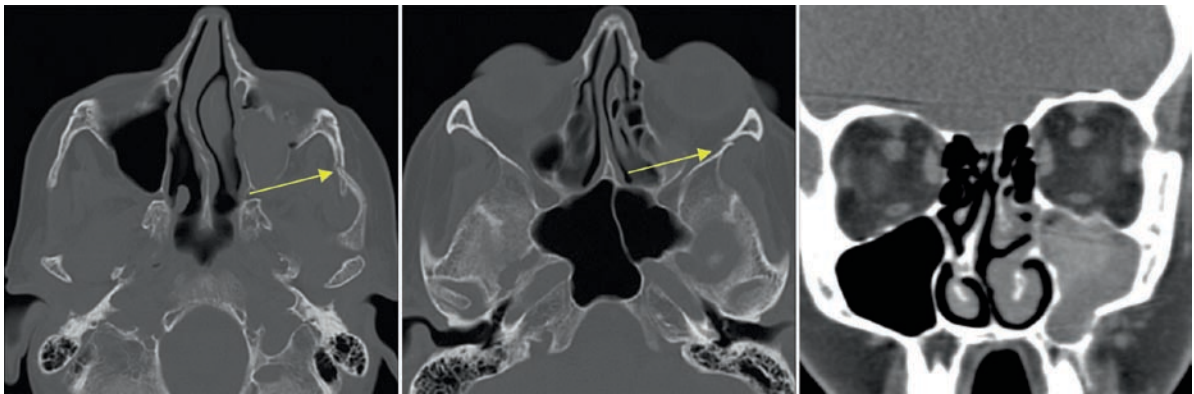


Fig. 3.8 Lateral midface fracture with posterior rotation of the zygomatic bone and fracture of the zygomatic arch and the lateral orbital wall (arrow). Hematosinus maxillaris on the left side

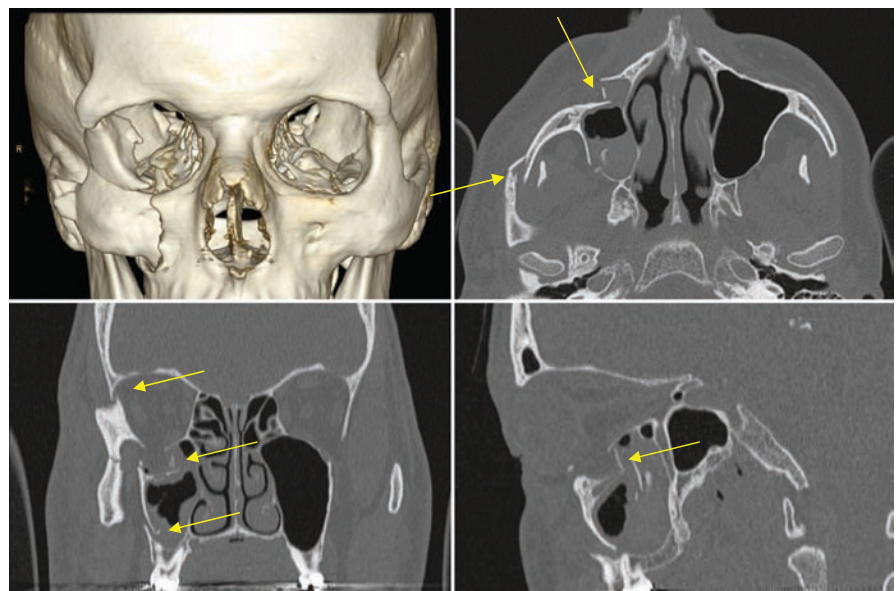
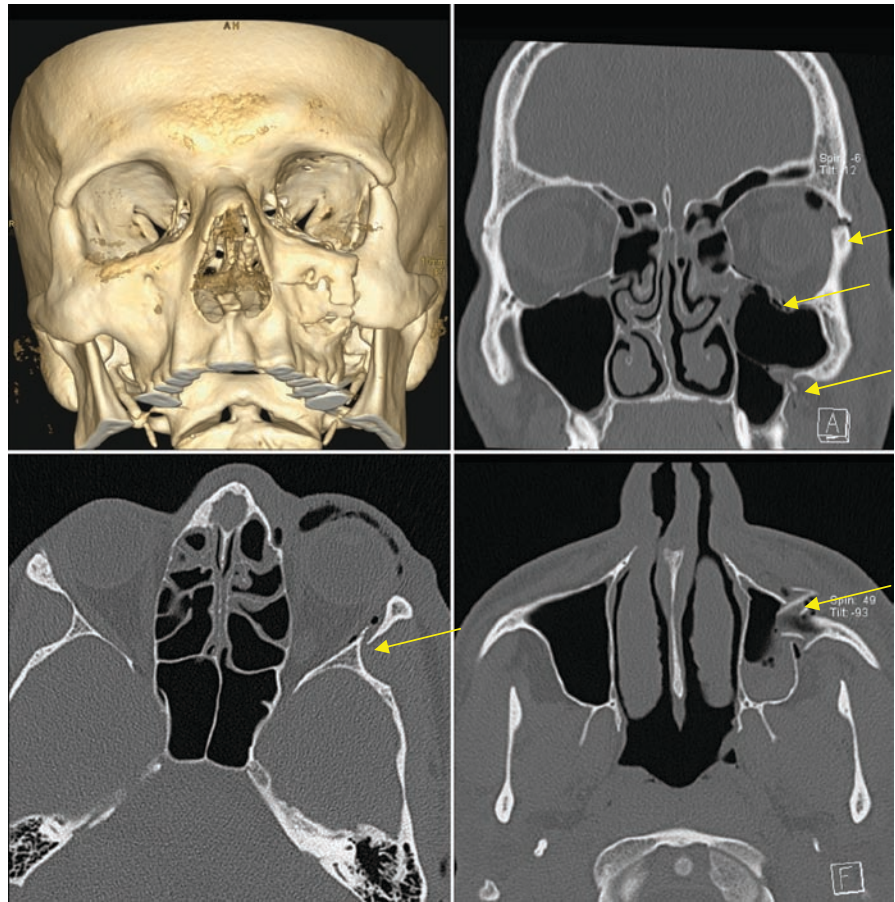


Fig. 3.9 Lateral midface impression fracture on the right side with marked medial and posterior dislocation of the zygomatic bone into the right maxillary sinus (arrow). Gross fragment of the lateral orbital wall. Marked depression of the anterior part of the orbital floor (arrow)

Fig. 3.10 Zygomatic fracture with medial and posterior dislocation (*arrow*). Volume reduction of the orbit. Fragmentation of the anterior wall of the maxillary sinus and orbital floor (*arrow*)



Fractures of the zygomatic complex may result in secondary fractures of the frontal bone, sphenoid bone, maxillary region or temporal bone, and as a result include the skull base in the fracture.

3.2.6 Midface: Combined Fractures

As the intensity of the impact kinetic energy increases, varying combinations of diverse fracture types may occur, with partly irregular progressions in differing fracture levels and midface compartments (Riefkohl et al. 1985; Mc Mahon et al. 2003).

With increasing comminution, there is a greater probability that the frontofacial compartment (IV) is involved with an immanent participation of the frontal skull base, frontal sinus and the ethmoidal cells (Boeninghaus 1974; Bull et al. 1989; Denneke et al. 1992; Weerda and Siegert 1992; Wolfe and Baker 1993; Stoll 1993; Schroeder 1993; Weerda 1995).

Fracture course: complex combined fractures

Here we are dealing with profoundly severe comminuted fractures of the midface as a result of brute force, which fails to comply with all rules and whereby there is almost always a simultaneous subcranial/intracranial trauma (Manson 1986, 1998). Besides open fractures, one finds impressions and asymmetries of the facial skeleton, there are often posttraumatic hypertelorism and dural injuries with rhinoliquorrhea with additional frontobasal fractures (Rowe and Williams 1985; Habal and Aryan 1989).

3.2.7 Naso-Orbito-Ethmoidal Fractures (NOE Fractures)

The interorbital skeleton is described as the NOE complex and is composed of a robust anterior section (nasal bone, frontal maxillary process) and a weaker dorsal

section [median skull base, ethmoidal cell system with crista galli, medial orbital walls (lacrimal bone-papyraceous plate, nasal orifices and conchae)] (Holt and Holt 1985; Messerklinger and Naumann 1995; Ewers et al. 1995; Weerda 1995; Prein et al. 1998; Schwenzer and Ehrenfeld 2002).

NOE fractures are difficult to classify as a result of their immense variability. Splinter fractures with totally random dislocations of varying fragment size are typical following injury to the interorbital region.

On midface impact with a hard object, the depressed naso-ethmoidal complex simultaneously involves the ethmoidal labyrinth, orbital walls, skull base and orbital soft tissues, and, due to the enlargement of the intercanthal distance, results in a posttraumatic telecanthus (Paskert and Manson 1988; Mathog et al. 1995).

Normal and pathologic distances of the naso-orbital complex (Holt and Holt 1985)

Normal intercanthal distance 30 mm
Normal interpupillary distance 60 mm
Telecanthus 45 mm

Today the classification from Markowitz et al. (1991) is generally accepted. It differentiates between three fracture types, which may appear uni- or bilaterally.

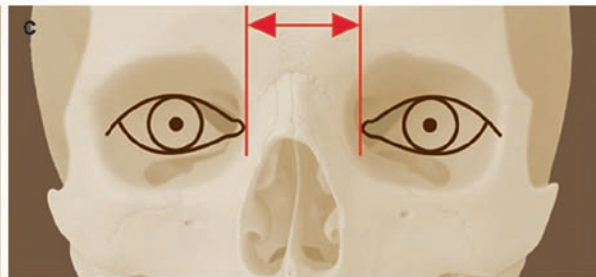
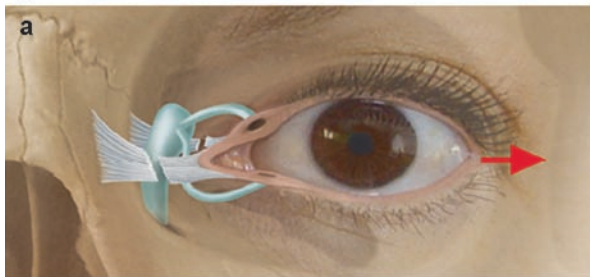
Classification of NOE fractures (Markowitz et al. 1991)

- Type 1 a: En bloc – fracture with moderate dislocation
- Type 1 b: En bloc – fracture with extensive dislocation
- Type 2: Multifragmental NOE complex with preservation of the canthal ligament attachment to central fragment
- Type 3: Multifragmental NOE complex with avulsion of the canthal ligament from the central fragment

The actual state of the *central* fragment is crucial, i.e., the region of bone bearing the medial ligamentous attachment of the lid (Hammer 1995; Hammer and Prein 1998).

One can differentiate between three types of injuries depending on the *severity of the cantho-ligamental displacement* (Mathog et al. 1995).

Type 1



Type 2

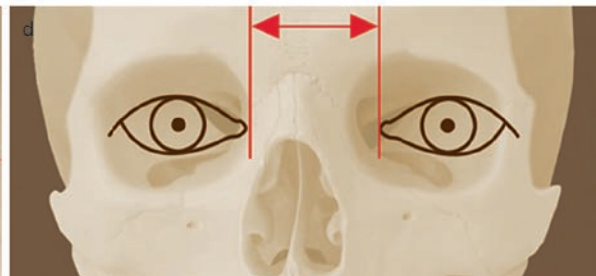
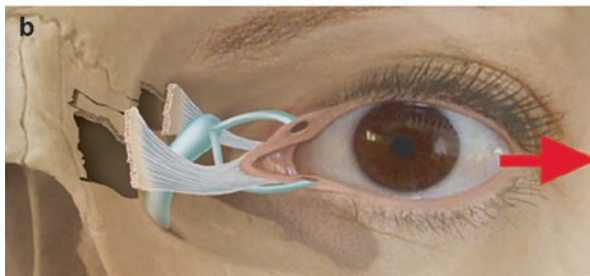


Fig. 3.11 Classification of the cantho-ligamental injuries in naso-orbito-ethmoidal fractures (NOE - fractures) (mod. a. MATHOG et al 1995) NOE fractures may be classified as type I, II, or III according to the severity of the injury (a-c) Type 1 – NOE - fracture with disruption of the medial canthal ligament and increase

in intercanthal distance without accompanying increase in interpupillary distance (posttraumatic telecanthus) (b-d) Type 2 – NOE - fracture with unilateral comminution and displacement of the medial orbital wall (central fragment) with adherent medial canthal ligament and accompanying increase in interpupillary distance



Fig. 3.12 Increase of intercanthal distance caused by a fracture of the right NOE complex. Enophthalmus caused by a right orbital floor fracture

Type 1: Avulsion of the medial canthal ligament
 Type 2: Unilateral osseous avulsion of the central bony region with adherent canthal ligament
 Type 3: Bilateral osseous avulsion of the central bony region with adherent canthal ligament (Fig. 3.11)

Canthal ligament injuries with disruption of the central fragment result in an increase in the intercanthal distance, due to the lateral position of the median canthi. The medial canthus is dislocated laterally; the curvature of the palpebral fissure vanishes through contraction of the orbicular muscle and is also laterally dislocated due to a laxity of the eye lids (Mathog 1992; Meleca and Mathog 1995; Mathog et al 1995). In type-3 NOE fractures, there is a comminution of the NOE complex with bilateral displacement of the medial orbital walls (central fragment) with adherent medial canthal ligaments. This results in flattening and widening of the naso-orbital complex and increase of the intercanthal distance (Figs. 3.12–3.17).

3.2.8 Cranio-Frontal Fractures

These fractures are staged according to the extent and dimension of the fracture and subdivided into the following fracture types (Bowerman 1985; Hoffman and Krause 1991; Mathog et al. 1995; Baker et al 2003):

- Isolated frontal sinus fractures
- Fractures of frontal sinus and ethmoid
- Fractures of frontal sinus, orbit, ethmoid, and bridge of the nose

Fractures of the craniofrontal region affect approximately 5% of all facial fractures and 2–12% of the cranial fractures (Ioannidis et al. 1993). In frontal sinus wall fractures there may be isolated or combined fractures of the anterior and posterior walls.

The varying fracture types can be differentiated with regard to their pathogenesis and surgical therapy (Godbersen and Kügelgen 1998):

Classification of frontal sinus fractures (Godbersen and Kügelgen 1998)

Type 1

Frontal sinus – anterior wall fracture
 Isolated or in combination with midface fractures

Type 2

Frontal sinus – posterior wall fracture without dura defect
 Isolated or in combination with anterior wall fracture and other midface fractures

Type 3

Frontal sinus – posterior wall fracture with dura defect
 Isolated or in combination with anterior wall fractures and other midface fractures (Figs. 3.18–3.20)

3.3. Craniofacial Fractures

3.3.1 Skull Base-Related Classification

In comparison with the past, injuries of the facial skeleton have changed considerably with reference to severity, extent, localization, and accident mechanisms, and tend toward a higher incidence of combined and severe facial- and cranial injuries (Manson 1986, 1998; Gruss et al. 1989; Hardt et al. 1990; Krafft et al. 1991; Schilli and Joss 1991; Vuillemin et al. 1998).

Craniofacial fractures are characterized by the inclusion of the frontofacial and frontobasal compartments in the midfacial fracture pattern. In many cases there are complex fractures with extensive comminutions and irregular fracture patterns as a result of maxillary, frontobasal, and frontofacial polyfragmentation (Matras and Kuderna 1980; Manson 1986, 1998; Schneider and Richter 1993; Weerda 1995; Hausamen and Schmelzeisen 1996; Joss et al. 2001) (Fig. 3.21).

Based on the modern industrial society of today and the decisively altered fracture mechanisms, Manson

Fig. 3.13 Fracture of the medial angle of the left orbit (frontal process of maxilla, lamina papyracea, medial infraorbital rim) (*arrows*) after direct trauma

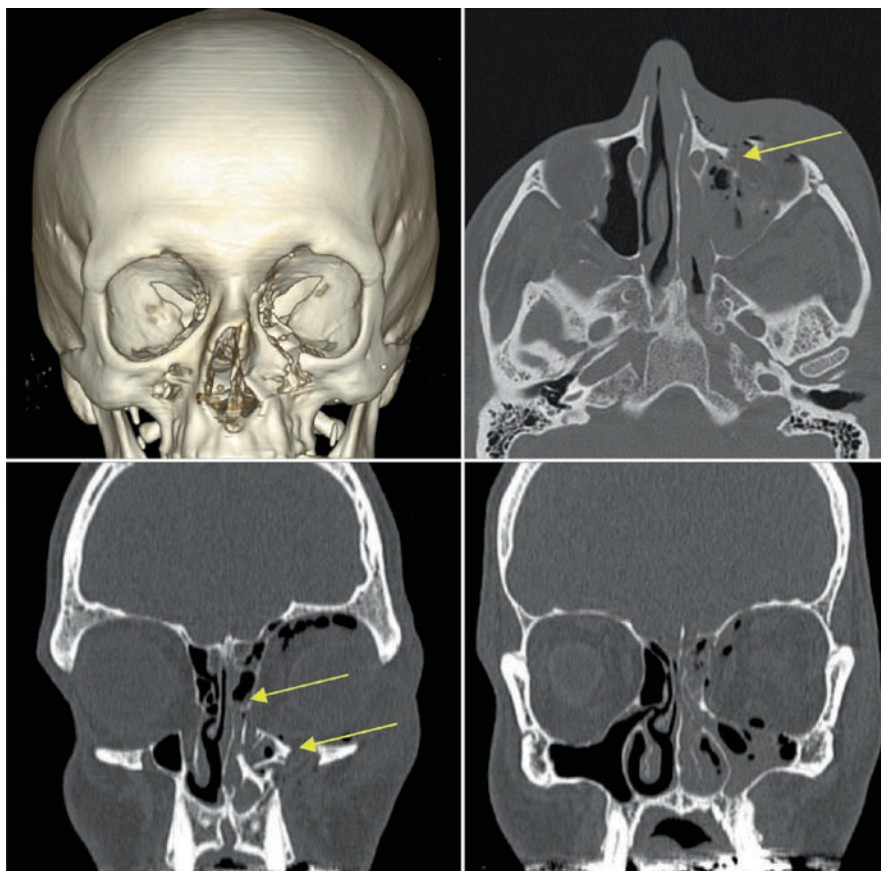


Fig. 3.14 Anterior naso-ethmoidal fracture with involvement of the cribriform plate (*arrows*)

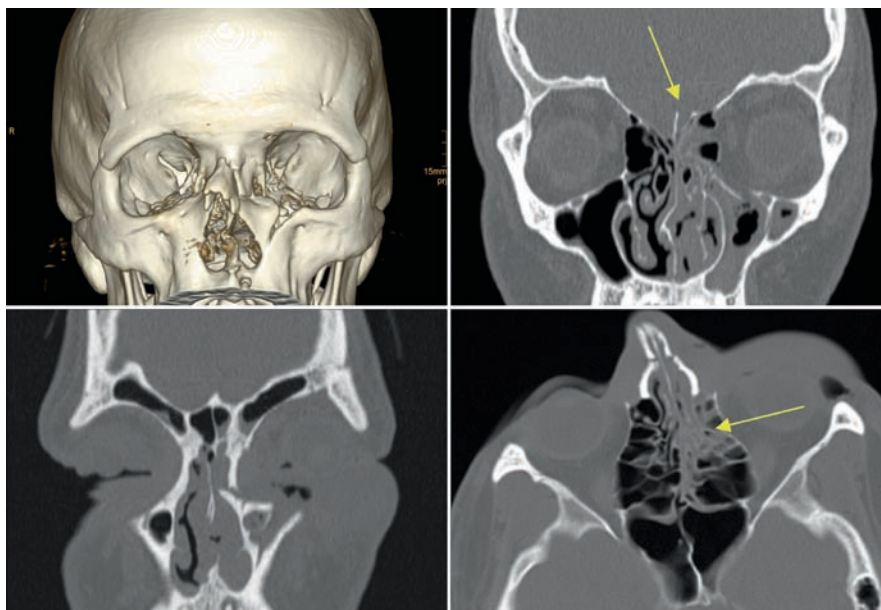


Fig. 3.15 Central midface fracture with posterior dislocation of the ethmoid (*arrows*) and fracture through the anterior wall of the frontal sinus and both maxillary sinuses

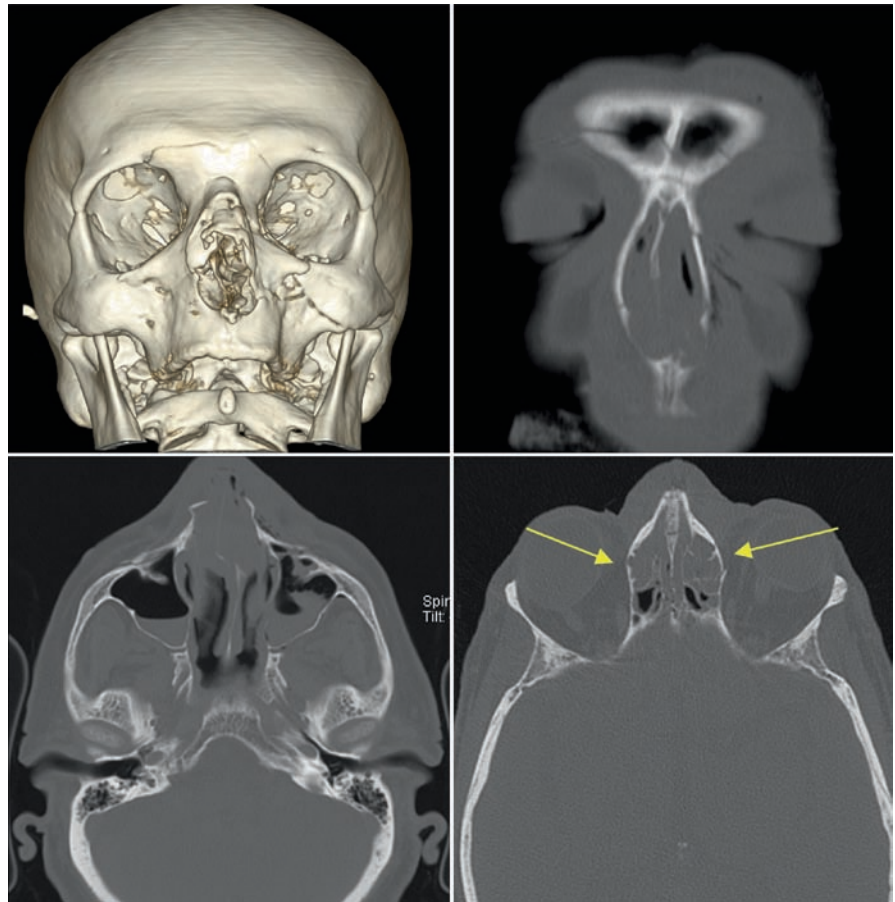


Fig. 3.16 Midface and naso-ethmoidal fracture with fragmentation of the posterior wall of the frontal sinus and posterior displacement of the nasal bone into the ethmoid (*arrow*)

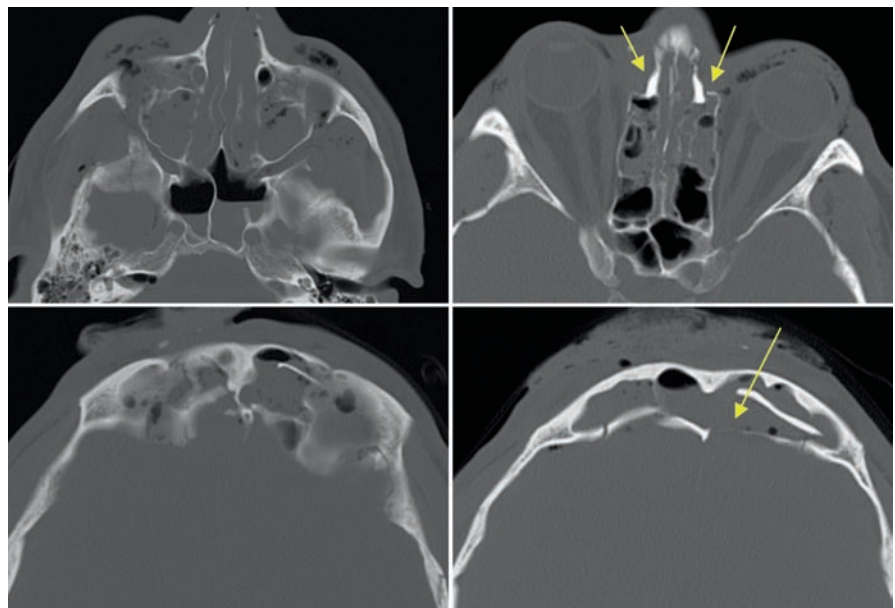


Fig. 3.17 NOE fracture with frontal skull base fracture (roof of the orbit, medial, and inferior orbital walls) (arrows)

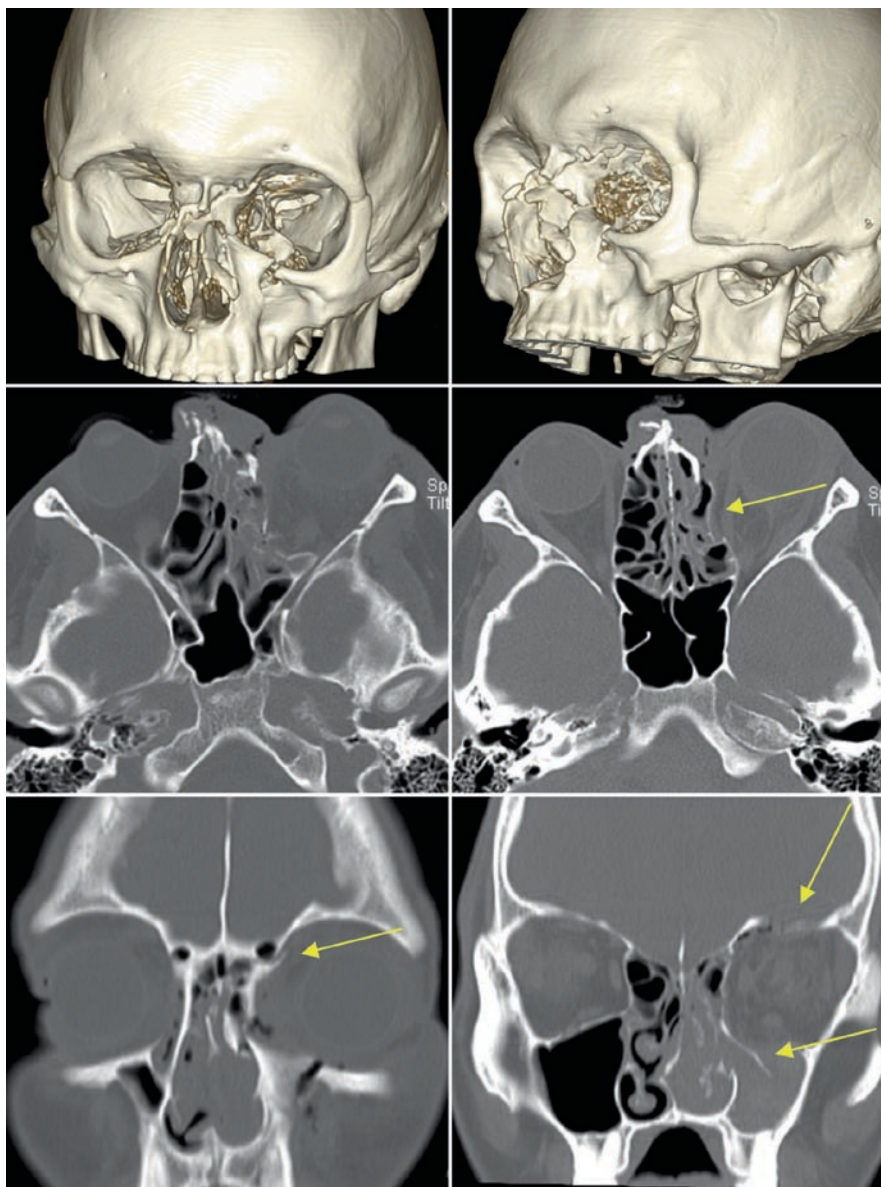


Fig. 3.18 Impression fracture of the anterior frontal sinus wall (arrow). The posterior wall is intact (type-1 fracture)

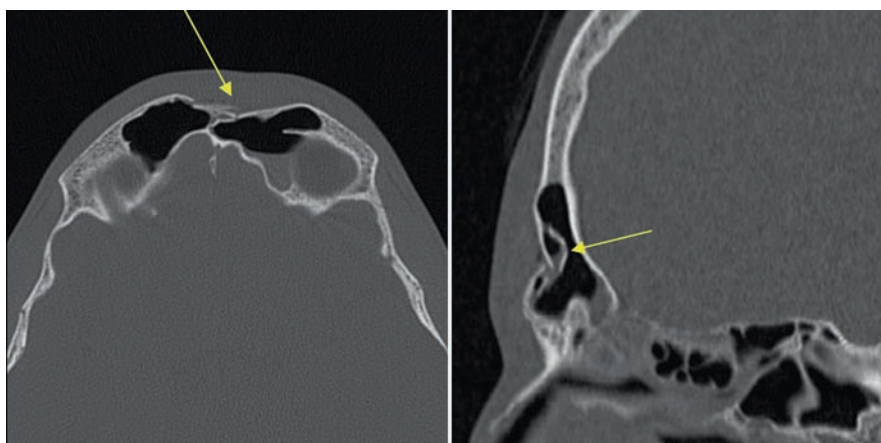


Fig. 3.19 Frontal impression fracture with contusion injury to left frontal lobe (*arrow*). Polyfragmentation of anterior and posterior wall of left frontal sinus (type-3 fracture) (*arrow*). Depression fracture of orbital roof

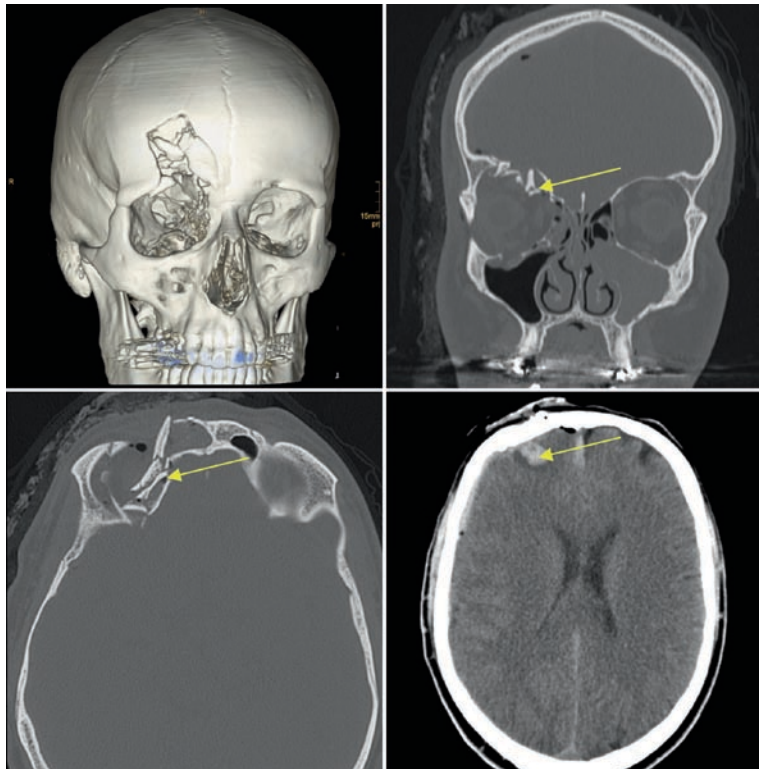
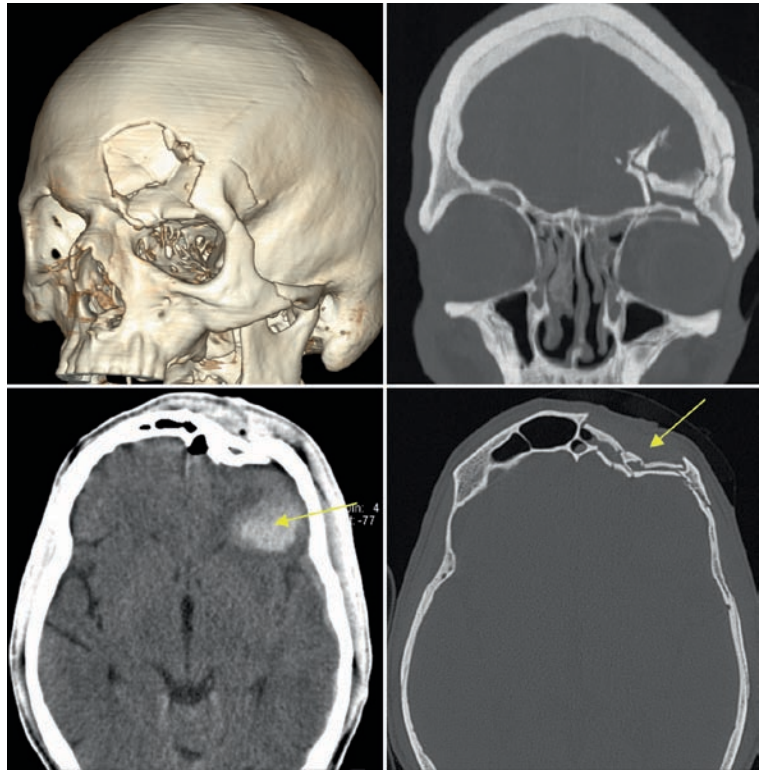


Fig. 3.20 Craniofrontal fracture after direct trauma (type-3 fracture). Polyfragmentation of the frontal sinus and orbital roof (*arrow*). Contusion injuries to the brain (*arrow*). Small amounts of air in the subdural space (d) indicating dural laceration

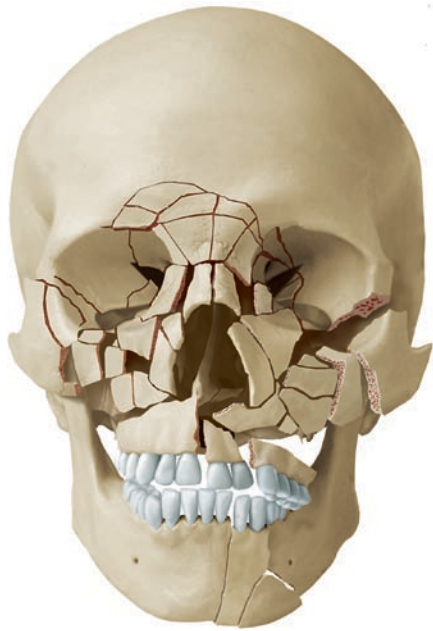


Fig. 3.21 Example of major craniofacial injury. Comminution and disruption of the subcranial midfacial skeleton including the naso-frontal region. Distocaudal dislocation of the midfacial complex with telescoping of the naso-fronto-ethmoidal region. Lateral dislocation of both zygomatic complexes. Bilateral dislocation of the nasal frame. Polyfragmentation of the anterior skull base, segmentation of the lower maxilla with dislocation of the alveolar segments. Paramedian fractures of the mandible

(1986, 1998) introduced a classification system according to *fracture dimension*, with a sub-division into *craniofacial* and *subcraniofacial* fractures. These fractures are referred to as comminuted (complex), to document the dimension and severity of the combined subcranial and craniofacial fractures.

- Complex subcranio-facial fractures (CUMF, CMF, LMF)
- Complex cranio-facial fractures (CCMF, PF)

The advantage of this classification is that the fronto-basal region forms the central reference point for craniofacial fractures, whereas in the old classification system it was the maxilla (Hardt et al. 1992).

3.3.2 Subcranial Facial Fractures

According to Manson (1986, 1998), the subcranial fractures of the Le Fort II, III, and Wassmund II, III, IV types are classified as:

- *Comminuted midface fractures (CMFs)*
- *Comminuted upper midface fractures (CUMFs)* — those fractures running beneath the skull base through the orbit and naso-ethmoidal region of the upper midface
- *Lower midface fractures (LMFs)* — the infrazygomatic fractures (Le Fort I fractures) (Figs. 3.22 and 3.23)

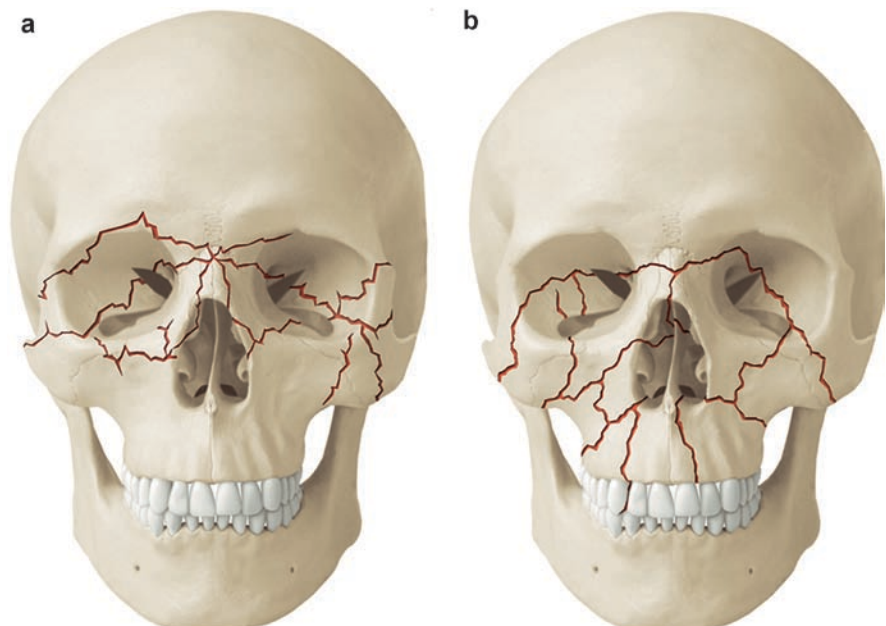
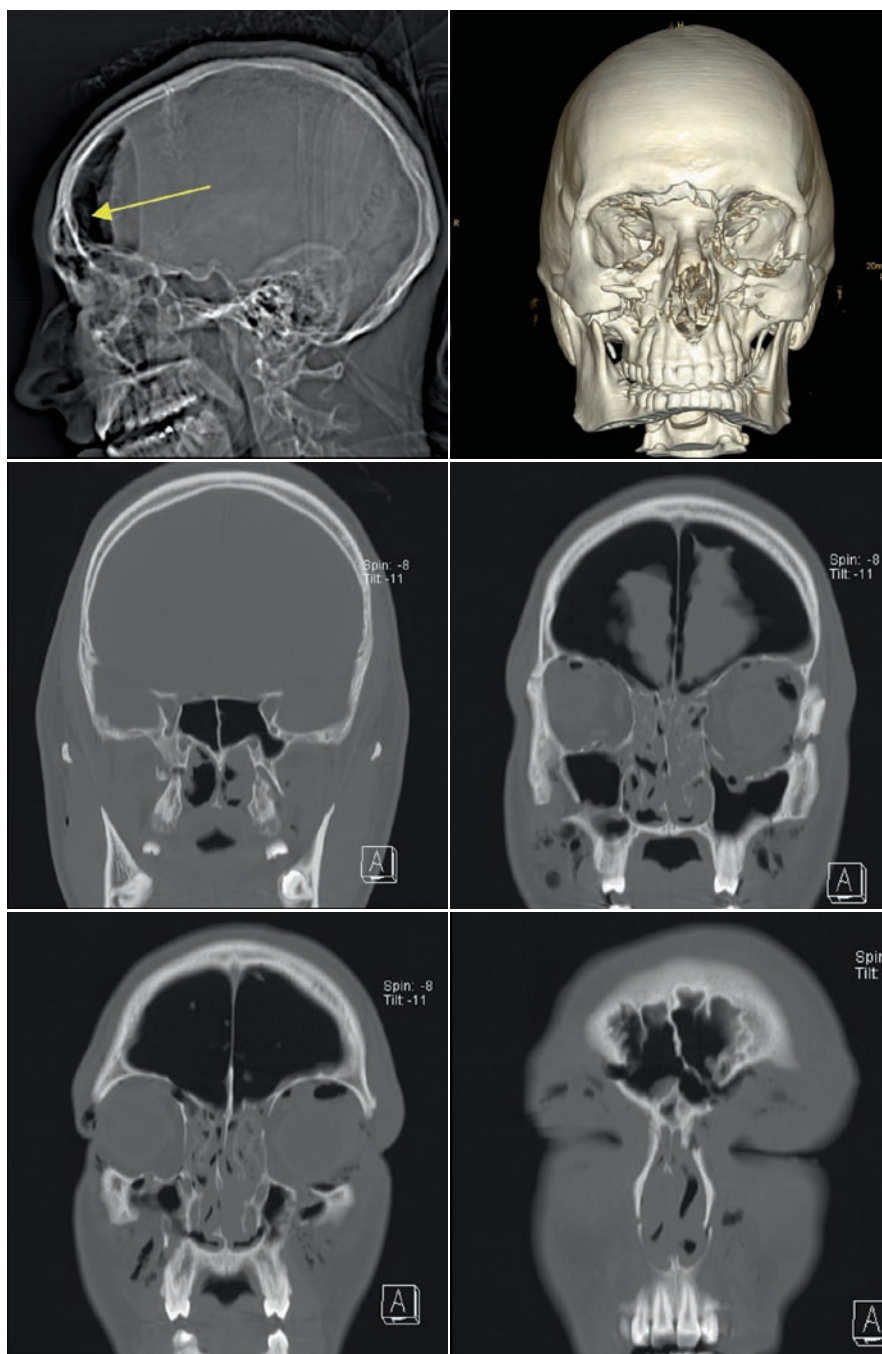


Fig. 3.22 Classification of complex subcranial/midface fractures (mod. a. Manson 1986, 1998). (a) Comminuted upper midface fracture (CUMF). (b) Comminuted midface fracture (CMF)

Fig. 3.23 Subcranial fractures with avulsion of the viscerocranium from the neurocranium. Frontobasal fracture with massive pneumocephalus (*arrow*) (LeFort I/II fracture, NOE and left zygomatico-orbital fracture) (CMF)



3.3.3 Craniofacial Fractures

Craniofacial fractures are subdivided into:

- Comminuted cranio-maxillary fractures (CCMFs)
 - a combination of differing cranio-maxillary fracture types with variable involvement of the orbital, naso-ethmoidal, and frontal regions
- Comminuted panfacial fractures (PFs)
 - which comprise the frontofacial region and the total facial skeleton

So, in *PFs* there is a combination of cranio-frontobasal fractures and fractures of the central and/or centro-lateral midface complex and the mandible, thus largely destroying the three-dimensional projection of the midface (Fig. 3.24).

CCMFs, which are confined to the cranio-fronto-orbital region, may be further subdivided into:

- Central *cranio-frontal fractures (CFFs)*
- Lateral *cranio-orbital fractures (COFs)*

according to the dimension of the fracture (Gruss 1982, 1985; Jackson et al. 1986) (Figs. 3.25 and 3.26).

3.3.4 Central Cranio-Frontal Fractures

The complex *CFFs* in the central frontal region affect the frontal sinus, the naso-ethmoidal complex and the neighboring orbital region either as isolated or combined fractures (Gross 1984; Donald 1998).

There may be an isolated or combined involvement of the anterior and posterior walls of the frontal sinus. As the lamellae of the posterior wall are significantly thinner than those of the anterior wall, a frontal stress-vector, with distortion of the skull through lateral tension can result in isolated bursting of the posterior wall (Hybels 1977) (Figs. 3.27 and 3.28).

3.3.5 Lateral Cranio-Orbital Fractures

The complex *COFs* comprise fractures of the lateral cranio-zygomatico-orbital region with variable participation of the orbital walls, anterolateral skull base, orbital roof and temporal region. Both orbital wall injuries and fractures of the orbital apex may result in neurovascular injuries (Fig. 3.29).

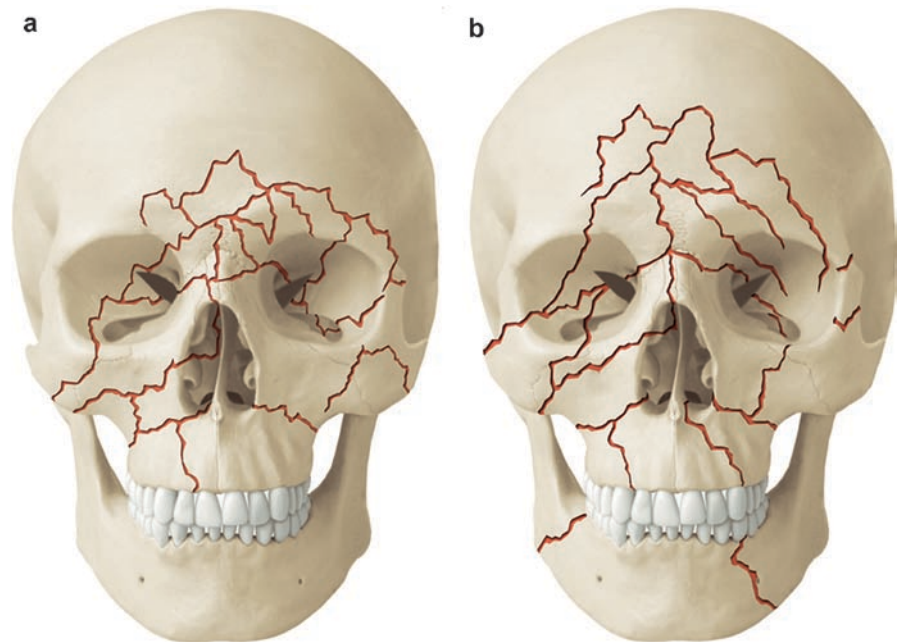


Fig. 3.24 Classification of complex craniofacial/midface fractures (mod. a. Manson 1986, 1998). (a) Comminuted cranio-maxillary fracture (CCMF). (b) Panfacial fracture (PF)

Fig. 3.25 Complex frontal skull and skull base fracture combined with naso-maxillary midface fracture (CCMF fracture) after oblique trauma across left frontal to right maxillary region. Comminuted fracture of the left orbito-frontal buttress (*arrow*). Flattening of the left frontal bone with underlying brain injury. Slight posterior dislocation of the naso-ethmoidal complex together with the large central fragment of the frontal bone (*arrow*)

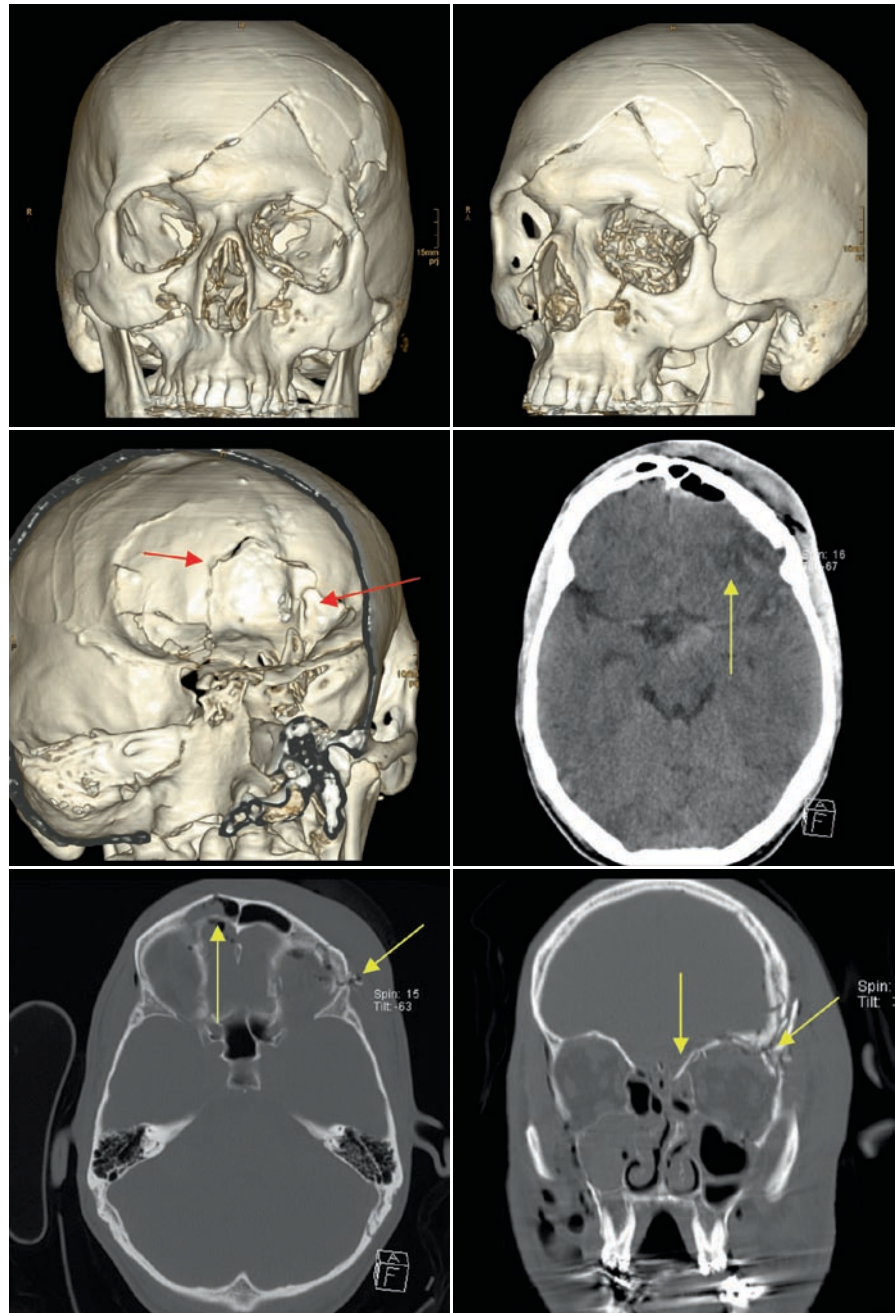


Fig. 3.26 Frontofacial and transorbital fracture (CCMF) with left temporal lobe injury after impact from the left side. Sagittal fracture through the orbit and frontal bone (*arrow*) with depression fracture of the orbital roof and zygomatic bone. Impression fracture of the fronto-nasal complex. Wide cleft in the medial part of the left frontal skull base (roof of orbit/ethmoid) (*arrow*)

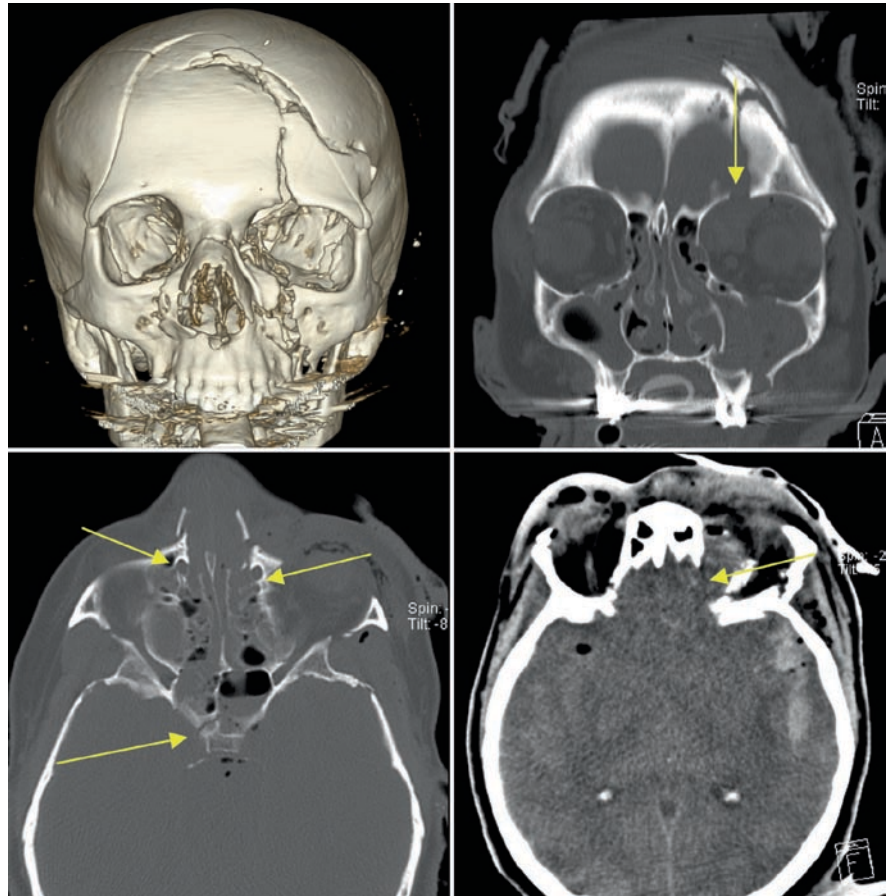


Fig. 3.27 Frontal impression fracture (CFF) with contusion injury to left frontal lobe (*arrow*). Polyfragmentation of the anterior and posterior wall of the left frontal sinus (*arrow*). Depression of the orbital roof (*arrow*)

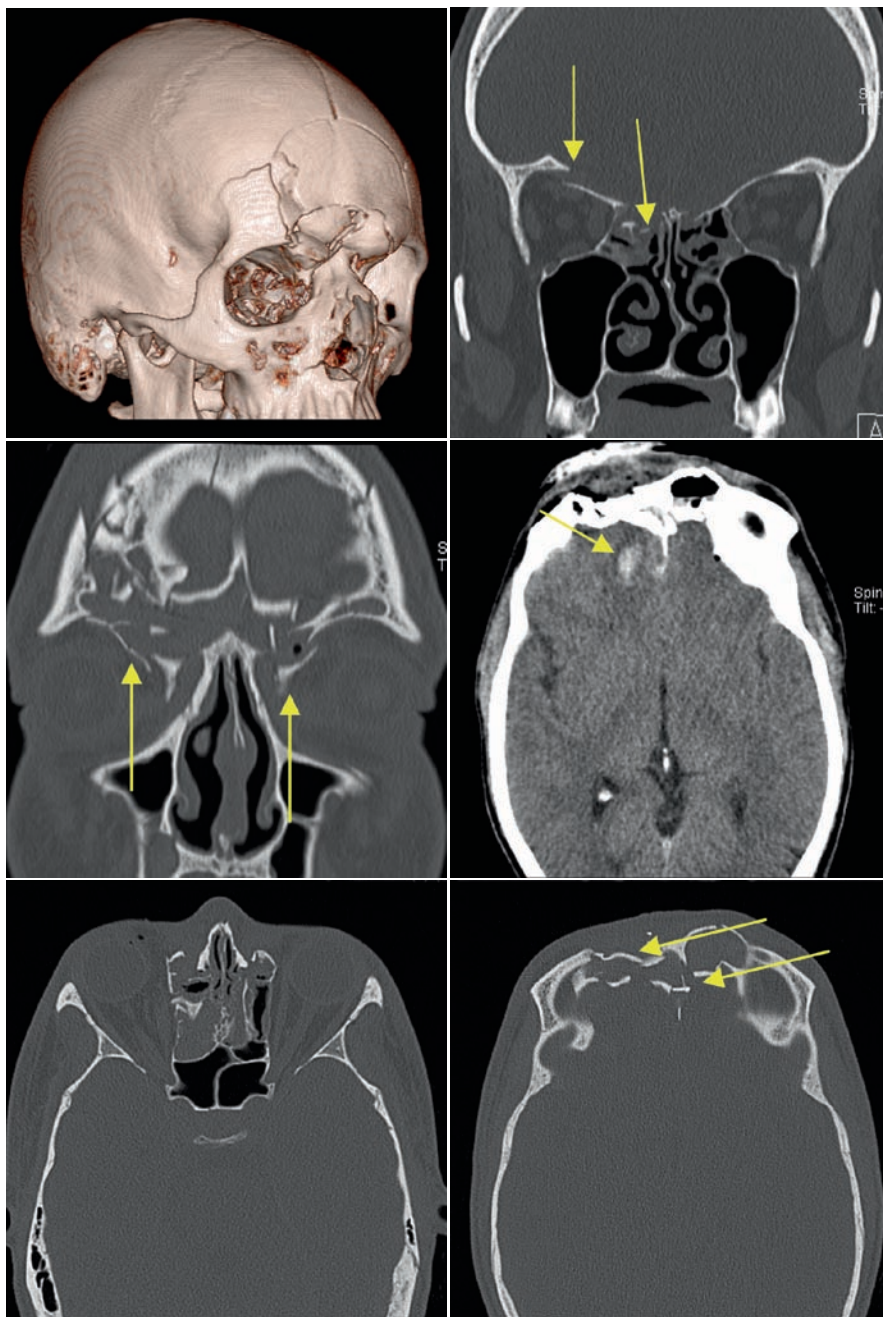
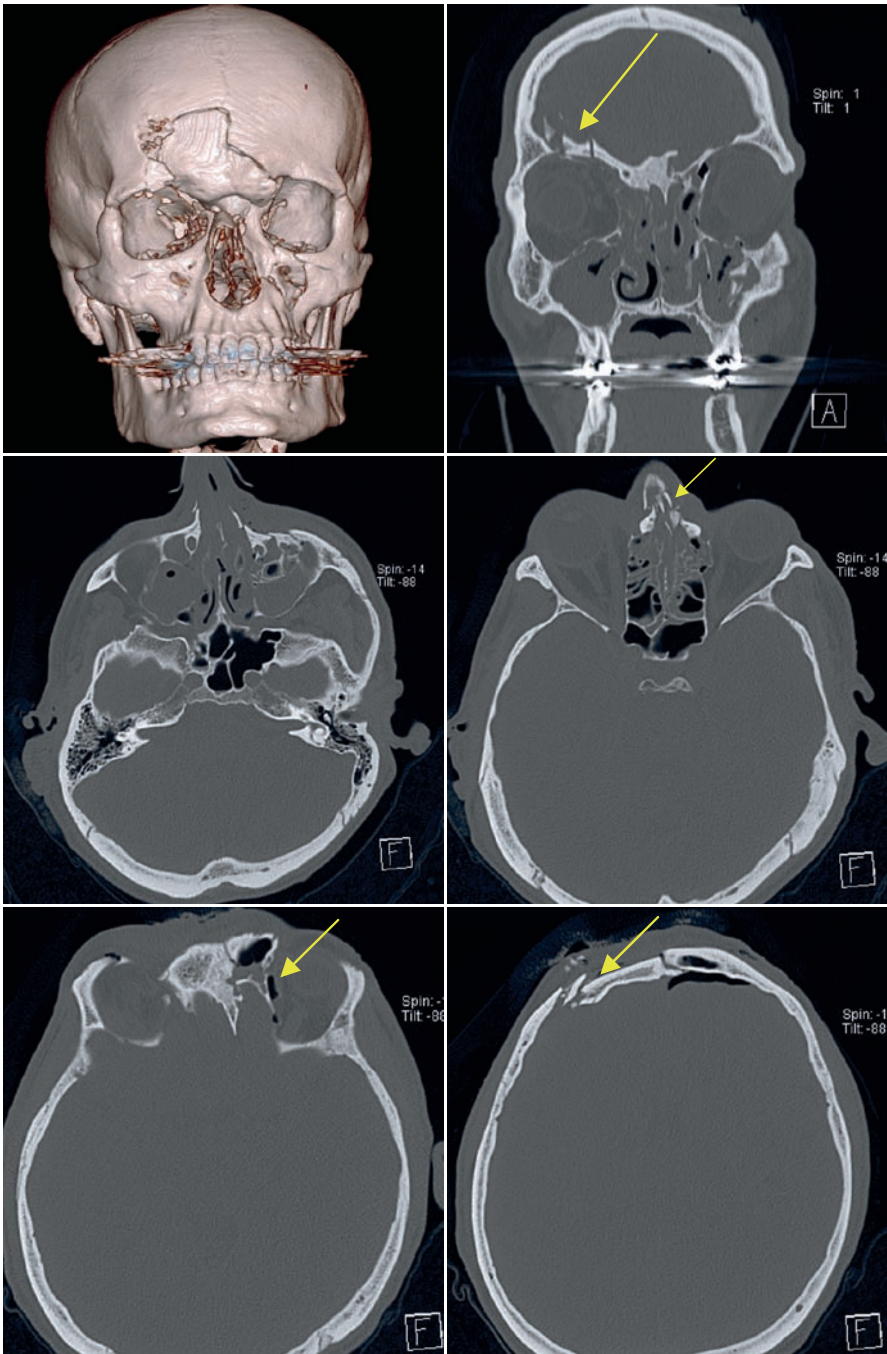


Fig. 3.28 Comminuted frontofacial fracture (CFF fracture) with polyfragmentation of the right frontal bone, orbital roof and frontal sinus (arrow) and posterior dislocation of the nasal bone



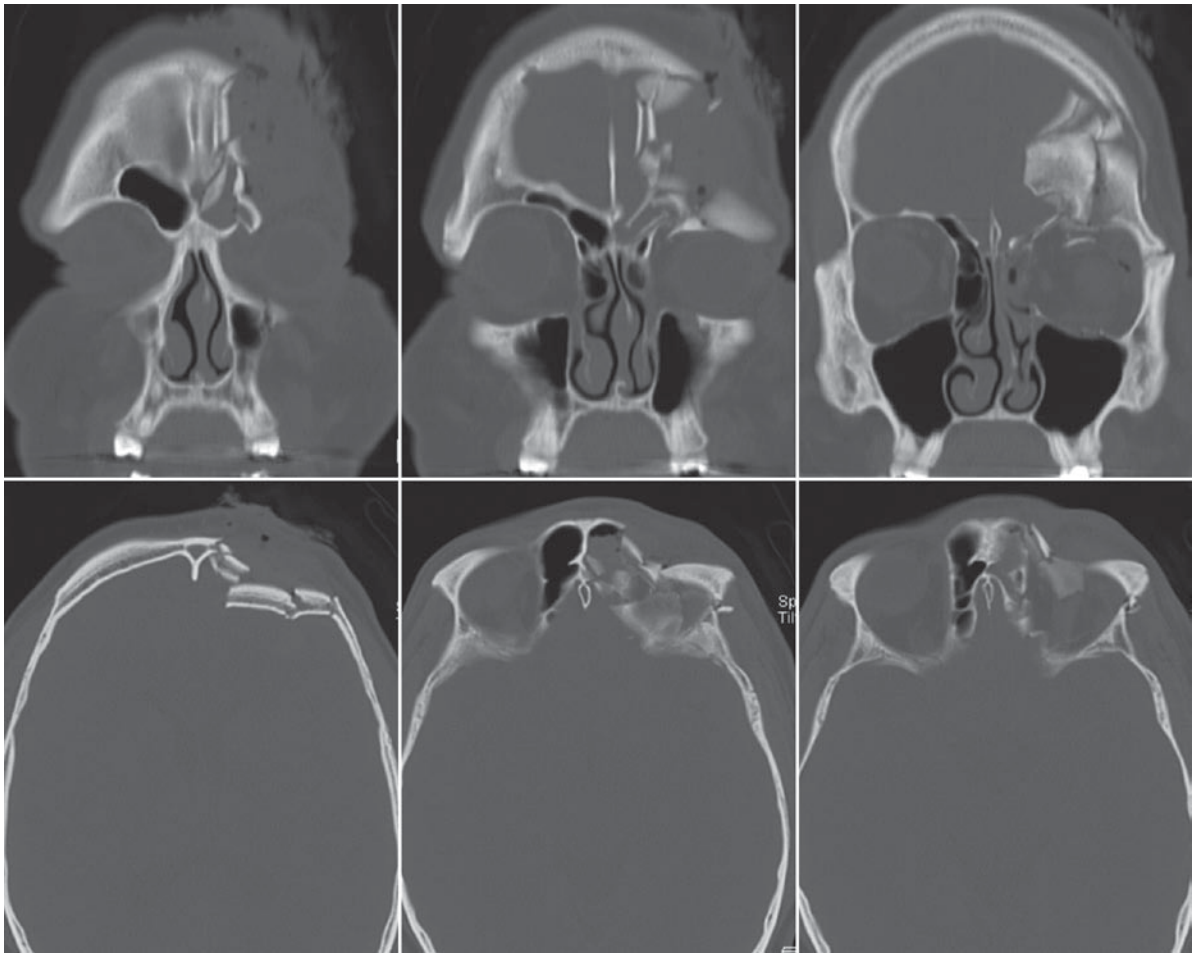


Fig. 3.29 Comminuted cranio-orbito-facial fracture (COF) with polyfragmentation of the left frontotemporal bone, orbital roof and frontotemporal base of the skull

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4.1 Fractures of the Skull Base

Longitudinal and transversal struts of the skull base form a framework of strong bone pillars similar to that existing in the face. The longitudinal strut starts at the central origin of the lesser wing of the sphenoid bone and runs through the middle and posterior cranial fossa, forming the foramen magnum, and tapers into the occipital bone. The two axes of the transversal strut run anteriorly through the region of the lesser wing of the sphenoid bone and posteriorly through the region of the petrous part of the temporal bone, forming ridges, which strengthen the skull base.

Fractures of the skull almost exclusively develop through deformation of the skull along established weak points (Kretschmer 1978). Depending on the direction and force of the impact, burst or bending fractures occur. The anatomical classification differentiates between skull fractures as each of these groups presents distinct clinical findings, specific aspects of management, risks, and complications.

Burst fractures, for instance, are typical fractures of the anterior skull base and bear the risk of brain injuries through bone fragments impinging on the brain.

4.1.1 Burst Fractures

A broad impact on the skull may cause comminuted fractures directly at the impact site or the energy may be dispersed and lead to a compression of the skull and skull base, developing comminuted fractures indirectly. The fine bone plates connecting the bony framework of the skull base are less elastic than the stronger bone struts. Thus, most skull base fractures

must be considered to be comminuted fractures. A transverse impact will result mostly in transverse fractures of the skull base, damaging the fifth and sixth cranial nerves where they emerge from the skull base. Longitudinal forces often result in comminuted fractures in the frontal base of the skull. Here, the optic and olfactory nerves are at risk to be damaged in the region of the optic canal and the roof of the ethmoidal cells (lamina cribrosa) (Probst 1971, 1986).

- Forces with a broad impact surface on the skull lead to compression, resulting in burst fractures of the skull base (Ernst et al. 2004)

4.1.2 Bending Fractures

Bending fractures occur preferably in the diploic bone architecture of the calvaria and are rarely seen in the skull base. A substantial force directed at a limited part of the skull bone can result in a typical impression fracture with the risk of brain damage or intracranial hematomas. High velocity impacts against the skull lead to comminuted fractures, blowout fractures, combined fractures of the skull and skull base or even fissures and bending fractures at the opposite side of the impact (Probst and Tomaschett 1990; Spangenberg et al. 1997).

- Forces with a small impact surface on the skullcap can lead either to fissure fractures or to bending fractures in the base area (Ernst et al. 2004)
- Forces with a small impact surface on the facial skeleton may lead to bending fractures of the frontal skull base with dislocation of bone fragments into the cranium

The convexity of the skull, the anatomical stability of the diploic calvarial bone and the skull base form

a strong spherical structure. The traumatic force is absorbed and dispersed. Thus, skull base fractures will lead to fracture lines extending into the calvaria or, vice versa, fractures of the calvaria extend into the skull base. However, fractures of the cranium normally do not continue into the midface region. Bending fractures in the frontal bone region typically process as impression fractures towards the region of the crista galli, the cribriform plate or ethmoidal bone and the orbital roof, thus directing traumatic energy to the facial skeleton.

4.2 Frontofacial: Frontobasal Fractures

4.2.1 Fracture Mechanism

Forces acting directly on the frontofacial skeleton lead to fractures of the thin bony plates in the frontal skull base. The structural difference in thickness and elasticity of the skull bone varies in each individual. The bony struts fracture or disrupt at the connecting sutures with the facial skeleton, whereas the thin bone plates of the frontal skull base will be splintered to fragments.

In frontofacial trauma, mainly the roof of the ethmoidal cells, the medial wall, and floor of the orbits are affected (Probst 1971; Probst and Tomaschett 1990). In 78% of the cases, the system of the paranasal sinuses close to the skull base is involved (Probst 1971, 1986). In severe trauma, the optic nerve canal and the sphenoid bone can be involved (Kessel et al. 1971; Probst 1971; Dutton and Al Qurainy 1991) Rohrbach et al. 2000).

In such a trauma the medial aspect of the sphenoid cap is exceptionally endangered. Here, the thin osseous plate of the lamella orbitalis of the sphenoid is connected with the cribriform plate and the orbital part of the frontal bone (Boenninghaus 1974; Probst 1986; Theissing 1996). In comparison with the thin bony structures of the orbital walls, the bony structure of the cribriform plate and the crista galli are relatively strong. This is the anatomical explanation for fracture lines encircling and dislocating the ethmoid complex “en bloc” (Probst 1971; Mathog 1992; Mathog et al. 1995).

This kind of injury mechanism is often seen in traffic accidents and extreme falls (Probst and Tomaschett 1990; Spangenberg et al. 1997), but only rarely in sport (Panzoni et al. 1983; Crow 1991; Gassner et al. 1999) or occupational accidents (Hill 1982; Hill et al. 1984).

A rare, but typical fracture mechanism leading to severe, life-threatening injuries occurs with boxing: the so-called “upper-cut.” A direct punch aimed at the chin or nose inferiorly can result in a fracture of the central part of the anterior skull base in the region of the cribriform plate with dislocation of bony fragments into the anterior cerebral fossa.

4.3 Midfacial: Frontobasal Fractures

The midface is defined as the connecting link between the frontobasal and maxillo-mandibular compartment. The aerated paranasal sinuses, the nasal passage and the orbits are energy absorbing structures protecting the skull base and the brain from direct trauma. Central and centro-lateral midface fractures can be associated with fractures of the walls of the frontal sinus. The frontal and/or posterior wall of the sinus may be affected.

High midface fractures can be associated with fractures of the frontal skull base. They occur as isolated complex nasofrontal fractures. The strong basis of the nose is dislocated into the anterior and middle part of the ethmoidal cells. Naso-orbito-ethmoidal (NOE) injuries with a severe impact on the high midfacial level may result in a dislocation of the crista galli and the cribriform plate into the anterior cranial cavity. This complicated injury is often seen in severe comminuted midface fractures (Lädrach et al. 1999).

- Fractures extend as direct bending fractures and as indirect burst fractures into the frontal skull base, the walls of the frontal sinus, into the frontal calvarial bones, but also towards the temporal bone

4.3.1 Trauma Factors

The severity of *skeletal midfacial injuries* is determined by a combination of various factors (Endo 1966; Bowerman 1985; Haskell 1985; Rowe and Williams 1985; Hardt et al. 1994). The localization, extent, and eventual damage to the visceral cranium depend on the impact force, the vector of the force, the impact point or contact area, and the level at which the injury is inflicted.

The potential damage is also determined by the form, surface structure and size of the injuring object, as well

as depending on the resistance and energy absorption of the skeletal structures, and the position of the head in the moment of the accident. Corresponding to the factors mentioned above, one can distinguish different patterns of injuries to the midface skeleton.

4.3.2 Impact Forces and Vectors

The skeleton of the midface is resistant to enormous forces approaching the midface region from below as the mandible absorbs a part of the traumatic energy.

Impact vectors that hit the midface from a *cranio-caudal* or *lateral* direction can lead to shear fractures dislocating parts of the midface or the complete midface complex.

Massive forces with an impact vector *directed horizontally* towards the midface result in comminuted fractures pushing the bony midface framework against the strong bone extensions of the skull base, e.g., the sphenoid bone. The whole midface complex can be separated from the skull base and dislocated en bloc backwards and downwards. The typical clinical appearance is called “dish-face deformity.”

Impact vectors directed at the infranasal region can result in fractures of the anterior alveolar process, but also in Le Fort I fractures, sometimes in combination with sagittal split fractures of the maxilla.

Impact vectors from a superior direction towards the upper part of the midface in most cases result in dislocated fractures of the NOE complex, if the head is in a normal position. This may be so if the contact surface is small, otherwise it might also lead to a shear fracture of the midface complex on the Le Fort III level.

Impact vectors from a lateral direction towards the midface typically lead to fractures of the zygomatic complex. Depending on the cranial or caudal direction of the vector a cranial rotation or caudal dislocation of the zygomatic complex may result. The zygomatic arch absorbs energy directed against the posterior aspect of the lateral midface. Depending on the energy applied, unilateral Le Fort II fractures may result in a combination of fractures of the orbital floor and the lateral alveolar process of the maxilla. Usually the lateral midface block rotates around a sagittal axis.

Horizontal impact forces may result in fractures of the lateral midface with extensions towards the nasomaxillary complex and the zygomatico-orbital complex.

The zygomatic arch may be broken down. Mild, but punctually directed forces can lead to isolated fractures of the homolateral alveolar process (Rowe and Williams 1985).

If the head is fixed at the moment of impact to the skull, laterally applied forces can result in fractures of the lateral skull bone in the region of the infratemporal fossa. As a rule, the zygomatico-maxillary complex is then impressed with a clear rotation around a vertical axis or central dislocation.

4.3.3 Structural Resistance and Energy Absorption

The impact energy is absorbed in different ways by the bony and soft tissue structures of the midface. The absorption is based on a variable bone resistance and strength as well as on the projection of the head at the moment of the impact (Nahum 1975; Hoffman and Krause 1991). A great variety of fracture patterns occur in the subcranial compartment related to the affecting kinetic energy (Rowe and Williams 1985; Bowerman 1985) (Fig. 4.1).

The following graphic illustration shows different absorption models. The magnitude of the kinetic energy applied has a direct influence on the fracture pattern, respectively to which extent the impact force can be absorbed by the midface complex (Fig. 4.2).

4.3.3.1 Degrees of Absorption

- Degree 1: A frontal impact with moderate force will result in multiple fractures of the prominent anatomical structures of the nasal complex and the midface. The central dislocation of fractured elements of the midface will absorb energy and helps to avoid skull base fractures
- Degree 2: In a frontal impact with considerable force, tear-off fractures and comminuted fractures of the anterior upper midface complex will occur, involving the naso-ethmoidal structures and sometimes the skull base
- Degree 3: Excessive force on frontal impact may result in comminuted fractures of the anterior and posterior upper midface complex

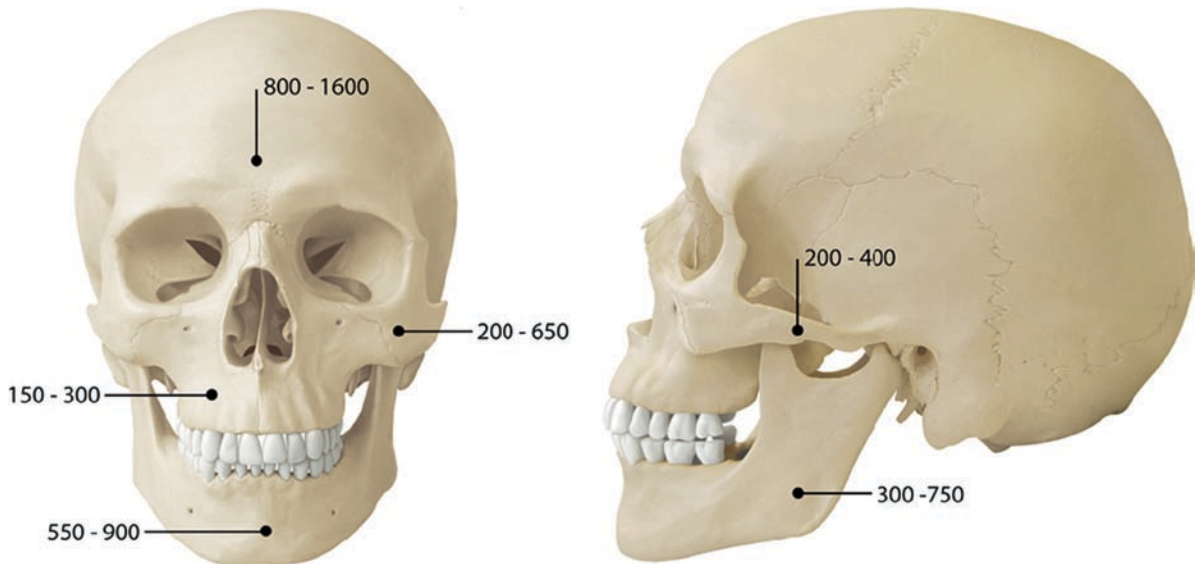


Fig. 4.1 Biomechanical forces (Nm) necessary to cause fractures of the facial skeleton at different locations. Note the differences between mandible, midface, and cranio-frontal region (mod. a. Nahum 1975; Hoffman and Krause 1991)

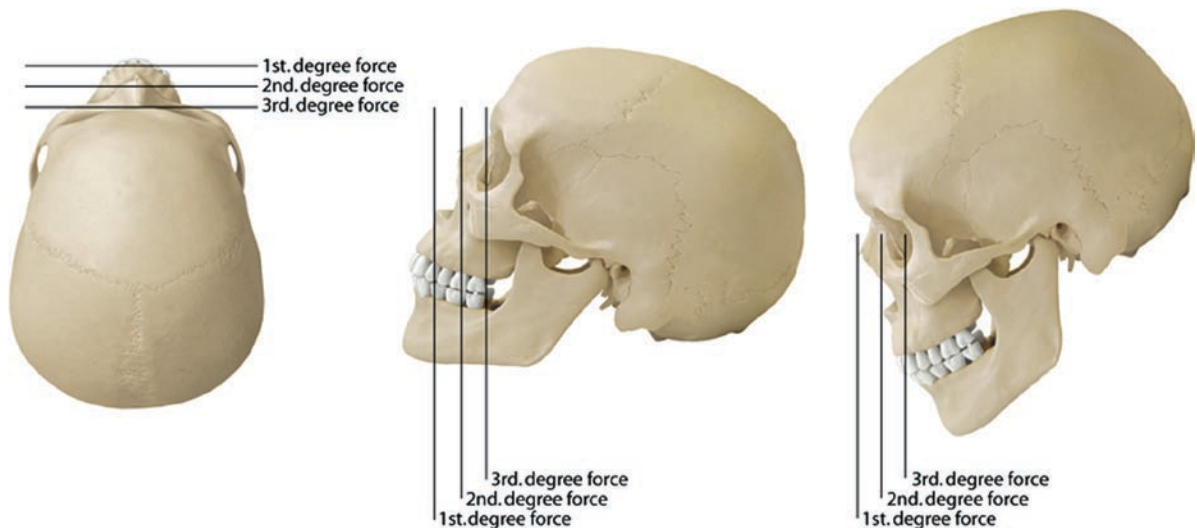


Fig. 4.2 Grading of the impaction force by extent of the midfacial absorption zones (degree 1-3). Location and depth of midface fracture zones in orthograd inclined and reclined head position (mod. a. Rowe and Williams 1985)

According to Ladrach et al. (1999), there are two different categories of complex cranio-orbito-maxillary fractures possible:

- Type I concerns a complex fracture that is limited to the external midface structure (no injury of the skull base)
- Type II (high-velocity trauma) concerns fractures of the external and internal midface structures, including

tear-off fractures of the midface complex, comminution of the bony midface framework and central dislocation of the anterior and posterior midface column with multi-fragmentary fractures of the skull base. Intracranial injuries may result. Type II fractures can lead to multiple dural lacerations, brain damage and herniation of the cerebral tissues into the paranasal sinuses or even the orbits (Fig. 4.3)

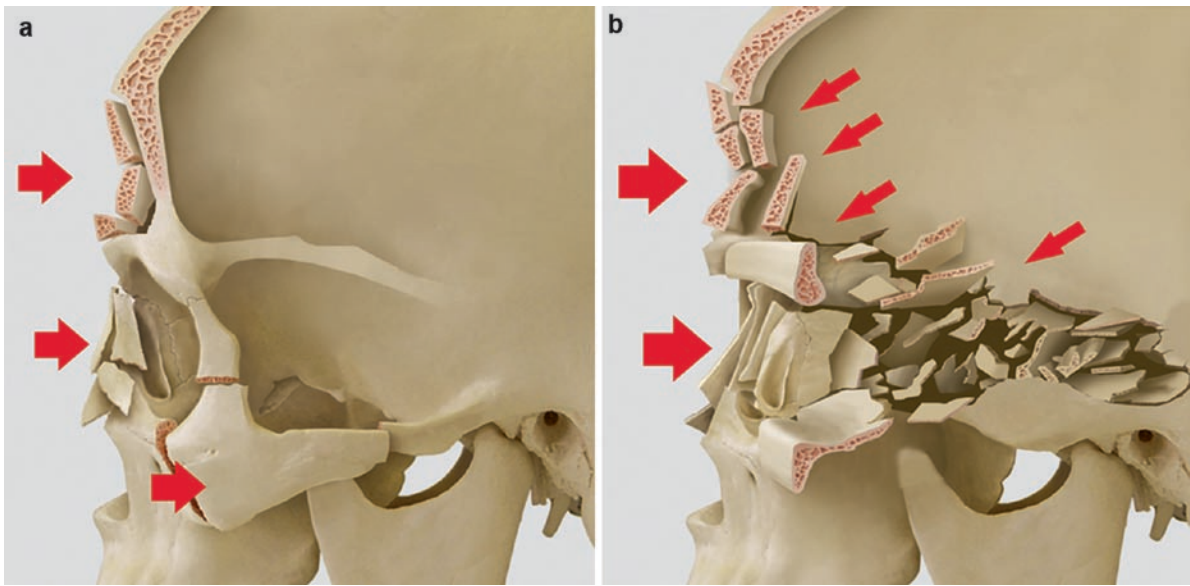


Fig. 4.3 Combined cranio-maxillo-orbital fractures. (a) Complex cranio-facial fracture limited to the external midfacial frame (type 1). (b) Complex cranio-facial fracture ("high velocity fracture pattern") with polyfragmentation of the anterior and posterior midfacial pillars, and comminuted basal fracture with dural and

intracranial injuries (type 2). The *small arrows* indicate the fractures of the cribriform plate and the ethmoid causing injury to the dura (mod. a. Lädach et al. 1999) (The *thick arrows* indicates the excessive force on frontal impact)

4.3.4 Impact Surface

A localized impact on a small, well-defined area of the facial skeleton will result in a different damage pattern than a traumatic force affecting a wide surface area of the face and skull. Small objects with minor velocity and energy will cause only limited injuries or fractures. The impact of a traumatic force with major velocity and energy acting on a large surface area, on the other hand, will result in extensive damage to the anatomical structures of the skull and face. Adjacent anatomical regions, such as the skull base or neurocranium, may be damaged as well.

4.3.4.1 Small Impact Surface

Objects with a diameter smaller than the diameter of the face will result in localized fractures of the naso-maxillary or dento-alveolar-premaxillary complex in the lower midfacial region; impacts in the central middle to upper part of the midface with moderate energy can lead to impression fractures of the naso-ethmoido-maxillary complex.

Fracture lines display a characteristic pattern and the central midface complex slides like a telescope into the interorbital cavity.

The impaction leads to a comminuted destruction of the thin osseous lamellae of the lacrimal bone and the ethmoid. The medial palpebral ligaments and periorbital soft tissues are regularly involved in such cases. The nasolacrimal ducts and the anterior skull base are frequently involved in the region of the cribriform plate or the orbital roofs.

An impact on the lateral region of the upper midface with limited energy and size will lead to fractures of the zygomatico-orbital complex.

4.3.4.2 Large Impact Surface

Large objects hitting the face and skull with a diameter larger than the diameter of the face or skull will result in an extended injury. The Le Fort fractures are typical results of these impacts.

Le Fort I fractures are caused by frontal and lateral impacts on the lower third of the midface. As a rule, the dislocation occurs in the direction of the acting force, mostly in a dorso-lateral direction; sometimes a

rotation of the maxilla round a vertical axis can be seen. Dorsal dislocation(s) as a result of traction of the pharyngeal muscles occurs rarely. However, it bears a high risk of airway obliteration. Only in atypical fracture patterns with disruption of the pterygoid process of the sphenoid can the pharyngeal muscular tract dislocate the fractured maxilla dorso-caudally.

Le Fort II fractures are caused by a blunt force that hits the middle part of the midface on a large impact surface. As the pterygoid process of the sphenoid is shorn off the skull base, the muscular pull of the medial pterygoid muscle can dislocate the midface dorso-caudally.

Le Fort III fractures are caused by blunt forces that hit the upper part of the midface on a large impact surface. As described above, a dorso-caudal dislocation of the fractured midface block is possible.

As the typical fracture line in Le Fort III fractures follows the plane of the skull base, additional fractures of the skull base may occur. Central and centrolateral fractures of the midface are often associated with fractures of the NOE complex (Gruss 1986; Probst and Tomaschett 1990; Kessler and Hardt 1998). NOE fractures are frequently combined with injuries to the skull base.

4.3.5 Position of the Skull

The damaging impact results from the energy affecting the object and the size of the object hitting the midface, but it also depends on the position of the head at the moment of impact.

4.3.5.1 Proclination

If the traumatic force hits the upper midface region in a proclined position of the head, the absorption by bony structures may prevent extended interorbital and skull base injuries, provided that the force is light to moderate (degree 1–2).

If the force is greater (degree 3), extended fractures of the NOE complex and of the glabellar, frontal, or supraorbital region are to be expected; these trauma patterns are due to a limited absorption zone frequently associated with skull base injuries (Rowe and Williams 1985).

4.3.5.2 Reclination

Considerable forces (degree 2) acting on the midface region in a reclined position of the head with the mouth open will lead to Le Fort II fractures. Extreme forces (degree 3) will result in Le Fort I or II fractures. Sagittal maxillary fractures with disruption of the median palatine suture can be expected.

If the force is primarily directed at the chin region, a mandibular fracture will occur with the risk of a bilateral condylar fracture. Subsequently the forces are absorbed by the midface complex leading to Le Fort I, II, and III fractures (Rowe and Williams 1985).

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5.1 Epidemiology

Traffic accidents are still the main cause of skull bone and skull base fractures. According to the literature, 40–70% of the casualties in traffic accidents suffer from multiple fractures in the viscer- and neuro-cranium (Kalsbeck et al. 1980; Crossman et al. 2003).

Sport accidents and accidents in leisure time follow, with 19%; the number of casualties in this group is increasing strongly (Probst 1971, 1986; Prokop 1980; Panzoni et al. 1983; Hill et al. 1984; Probst and Tomaschett 1990; Spangenberg et al. 1997; Gassner et al. 1999).

Skiing, biking and horse riding are the main activities with a high accident risk for cranio-maxillofacial injuries (Haeusler 1975; Crow 1991).

In 41% of our own patients, traffic accidents were the main cause of craniofacial trauma. Amongst them, 18% were due to car accidents, 17% due to bike accidents and 6% due to motorbike accidents. Falling during domestic activities caused approximately 23% of

the craniofacial injuries. Alcohol plays an important role in domestic accidents.

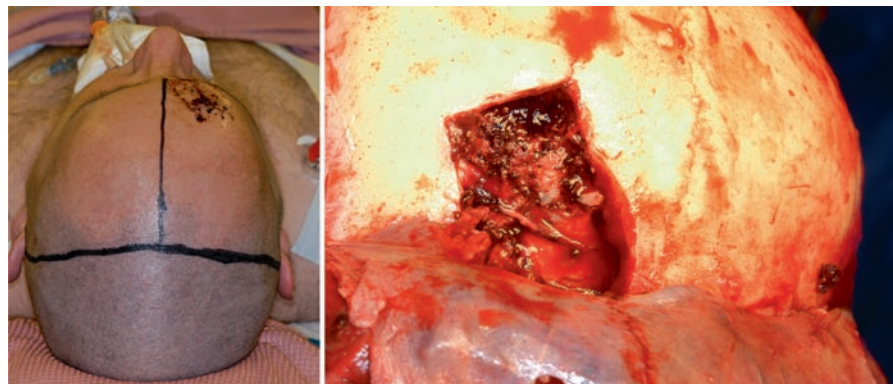
Sport activities were the cause in 18% of the craniofacial injuries. Ten percent of the craniofacial injuries were acquired at work. In 8%, violence was the cause, whereas 6% of the craniofacial injuries were related to suicide attempts using firearms and shotguns (Neidhardt 2002) (Fig. 5.1).

Statistical analysis of craniofacial fractures in our patients (Neidhardt 2002)

Traffic	41%
Domestic accidents	23%
Sports	18%
Work related	10%
Violence	8%

In conclusion, statistically in about 90% of the cases traffic and sporting accidents, as well as falling and work-related accidents, are responsible for serious and often multiple fractures in the frontofacial and fronto-basal part of the visceral and neural cranium.

Fig. 5.1 Impression fracture of the right frontal bone with injury to the dura and brain (skiing accident)



5.2 Skull Base Fractures/Meningeal Injuries

Nearly 5–20% of all cranio-cerebral injuries (CCIs) are associated with skull base fractures (Dietz 1970a, b; Loew et al. 1984; Ommaya 1985; Fournier 2007).

Depending on the trauma mechanism, one can distinguish open and closed cranio-cerebral traumas. If the dura mater is intact, the injury is defined as a closed or “covered” injury. If there is a laceration of the meninges or the sinus system, one speaks of an open brain lesion. An open brain lesion, caused by penetration or by tearing of the meninges, results in a liquor fistula (Schaller 2003).

5.2.1 Frequency

The literature states that in 3–11% of the anterior skull base fractures there are additional meningeal lesions with subsequent loss of cerebro-spinal fluid (CSF leakage) (Boenninghaus 1971; Ommaya 1985; Dagi and George 1988; Schmidek and Sweet 1988; Schroth et al. 1998, 2004).

The collateral swelling can obliterate an existing dura laceration in skull base injuries. In this case, a primary loss of cerebro-spinal fluid is clinically nondetectable (Ernst et al. 2004). One can overlook the loss of cerebro-spinal fluid due to traumatic obliteration by blood clots, bone fragments or by trapped brain tissues (Dietz 1970a, b; Strohecker 1984; Probst and Tomaschett 1990).

With 18% vs 86% there is statistical evidence for a striking discrepancy between the immediately clinically evident meningeal injuries and the intraoperatively detected actual meningeal lacerations (Dietz 1970a, b; Strohecker 1984; Dietrich et al. 1993; Kral et al. 1997 (96% -intraoperative). Considering the fact that patients with serious cranio-cerebral trauma are often admitted when intubated, it is very difficult to clinically detect a cerebro-spinal leakage in the emergency room.

5.2.2 Localization

A disruption of the meningeal tissues is most likely in the anterior cranial fossa where the dura is rigidly fixed to the cribriform plate, in the posterior wall of the frontal sinus and the posterior part of the roof of the ethmoid bone.

The meningeal tissues are also very vulnerable at the rigid dura attachment at the top of the sphenoid sinus and at the temporal part of the roof of the orbit (Kretschmer 1978; Ernst et al. 2004).

Isolated skull base fractures in combination with dural injuries occur most often in the region of the ethmoid and cribriform plate, followed by fractures of the orbital roof and the posterior wall of the frontal sinus (Probst 1986; Probst and Tomaschett 1990; Kocks 1993).

Depending on the severity and the extension of the cranial injury, multiple dura disruptions can occur.

Localization of skull base fractures in cerebro-cranial trauma (Probst 1986)

Ethmoid and cribriform plate	53%
Roof of the orbit	27%
Posterior sinus wall	17%
Sphenoid bone	3%

5.3 Midface: Skull Base Fractures

A statistical survey from craniofacial trauma centers presents extensive evidence of combined midface and skull base fractures depending on the severity and extension of the injury (Hausamen and Schmidseider 1975; O Brian and Reade 1984; Jacobs 1984; Manson et al. 1987; Brachvogel et al. 1991; Schilli and Joss 1991; Wahlmann and Wagner 1991; Haug et al. 1992; Denneke et al. 1992; Hardt et al. 1990; Raveh et al. 1992; Schroeder 1993; Weerden 1995; Hausamen and Schmelzeisen 1996; Koch and Lehnhardt 2000; Joos et al. 2001; Mc Mahon et al. 2003).

- Skull base fracture diastasis in craniofacial fractures

Vajda et al. (1987) provided CT data on bone diastasis in different craniofacial fractures with dural injuries. They observed a diastasis of more than 6 mm in high midface/skull fractures (ESCHER type I) and a diastasis of less than 5 mm in central (cribriform plate, posterior ethmoid) midface fractures (ESCHER type II). In ESCHER type III fractures with disruption of the midface from the skull base, there was a median diastasis of 4.8 mm. In all combined midface and frontal skull base fractures, a mean bone dislocation of 5.7 mm, with a range of 3.2–12.8 mm, was found.

- Skull base fracture frequency in craniofacial fractures

The number of skull base fractures associated with complex midface fractures is significantly higher in

comparison with skull base fractures associated with a cranio-cerebral trauma.

Frequency of skull base fractures in isolated cranio-cerebral trauma and skull base fractures in complex midface fractures

Skull base fractures in isolated cranio-cerebral trauma (Dagi and George 1988)	21%
Skull base fractures in complex midface fractures (O Brian and Reade 1984)	43%

5.3.1 Involvement of the Skull Base in High Midface Fractures

The involvement of the skull base in subcranial midface fractures varies, depending on the type of midface fracture and the severity of the injury. In 25–46% of Le Fort II and Le Fort III fractures, further skull base fractures can be expected (Waller 1977; Vajda et al. 1987; Neidhardt 2002).

With reference to the Le Fort classifications, the frequency of skull base fractures in subcranial midface fractures can be subdivided as follows:

Skull base fractures in midface fractures (Le Fort type fracture) (Neidhardt 2002)

Le Fort I fractures	1%
Le Fort II fractures	37%
Le Fort III fractures	10%
Combined Le Fort I, II, III fractures	52%

According to our own data, midface fractures of the central compartment of the midface are related to skull base fractures in up to 62% of the cases. Centrolateral midface fractures occur in 25%; lateral midface fractures in 13% (Hardt et al. 1990; Neidhardt 2002).

These results are similar to the data published by Raveh and Vuillemin in 1988, who found an involvement of the skull base in 54% in central midface fractures and 12% in lateral midface fractures.

Skull base fractures in subcranial midface fractures (Neidhardt 2002)

Central midface fractures	62%
Centrolateral midface fractures	25%
Lateral midface fractures	13%

Skull base injuries predominate in complex craniofacial traumas (Hardt et al. 1990). Out of 268 elective serious midface injuries presented to the Department of Oral and Maxillofacial Surgery of the Kantonsspital

Lucerne (1980–2005), 73% had additional injuries to the frontofacial skeleton. Of these injuries, 67% were termed craniofacial and 33% subcranial fractures.

In the severe *craniofacial fracture* group, fractures of the skull base occurred in 45% of the cases [comminuted cranio-midface fractures (CCMFs)]; 22% of the casualties were termed panfacial fractures (PFs).

In the subgroup with *subcranial fractures*, skull base fractures were seen in 21% of the patients [comminuted upper midface fractures (CUMFs)] and in 12% comminuted midface fractures were diagnosed [comminuted midface fractures (CMFs)] (Hardt et al. 1990; Neidhardt 2002).

Involvement of the frontobasal compartment in subcranial and craniofacial fractures (Neidhardt 2002)

<i>Craniofacial fractures</i>	67%
CCMFs	45%
PFs	22%
<i>Subcranial fractures</i>	33%
CUMFs	21%
CMFs	12%

5.3.2 Dural Injuries

5.3.2.1 Frequency

The frequency of simultaneous dural injuries in midface fractures varies, depending on the severity and extension of the osseous lesions.

According to the literature, between 41 and 70% of the *craniofacial fractures* are associated with dural injuries [Manson et al. 1987 (50–70%); Vajda et al. 1987 (41%); Hausamen and Schmidseider 1975 (44%); Raveh and Vuillemin 1988 (70%); Neidhardt 2002 (56%)].

In 18–31%, typical *subcranial midface fractures* are associated with simultaneous dural injuries [Waller 1977 (25%); Manson et al. 1987 (26%); Vajda et al. 1987 (18%); Brachvogel et al. 1991 (31%); Neidhardt 2002 (20%)] and 31% of the casualties with *craniofrontal fractures* [CFFs, including cranio-orbital fractures (COFs)] suffer from dural lacerations (Neidhardt 2002).

Frequency of dural injuries in complex craniofacial, craniofrontal, and subcranial midface fractures (Neidhardt 2002)

Craniofacial fracture	CCMF/PF	56%
Craniofrontal fracture	COF/CFF	31%
Subcranial fracture	CUMF/CMF	20%

The trabecular skeletal framework of the midface absorbs a great deal of the kinetic energy delivered by an accidental impact. This absorbing function of the strong bony framework surrounding the facial cavities avoids direct energy transfer towards the skull base, the endocranium or the eyeball. Impacts hitting the lower midface are rarely combined with skull base fractures or dura lacerations (Vajda et al. 1987).

In conclusion, dural injuries are more common in craniofacial fractures than in subcranial fractures. In general, one has to bear in mind that in about 50% of the patients with serious midface fractures the skull base and the dura may be involved (O Brian and Reade 1984; Gruss 1986; Probst and Tomaschett 1990; Hardt et al. 1990; Kessler and Hardt 1998).

5.3.2.2 Localization

Forty-seven percent of our patients showed dural injuries in the region of the ethmoid roof and the cribriform plate. The orbital roof was involved in 24% of the cases and the posterior wall of the sinus in 27% (Hardt et al. 1990; Neidhardt 2002).

Localization of frontobasal-dural injuries in craniofacial fractures (Neidhardt 2002)

Ethmoid and cribriform plate	47%
Posterior sinus wall	27%
Roof of the orbit	24%
Sphenoid	4%

Other studies on localization of cranio-facial/skull base fractures confirm these data (Raveh et al. 1998).

Localization of frontobasal-dural injuries in craniofacial fractures (Raveh et al. 1998)

Roof of the orbit/ethmoid/posterior sinus wall	68%
Cribriform plate	9%
Sphenoid/sella	3%

In 40–65% of the cases of both craniofacial and subcranial midface fractures, dural injuries occur mostly in the region of the cribriform plate and the roof of the ethmoid. Of dural lacerations, 15–30% occur isolated in the cribriform plate; in about 15% of the cases, only the ethmoidal roof or the posterior wall of the frontal sinus is involved. Between 20 and 30% of these fractures run through the orbital roof. In 3–9%, the region of the sphenoidal sinus is involved.

5.4 Cranio-Fronto-Ethmoidal Fractures

Isolated frontal sinus fractures afflict the anterior wall in 29%, the posterior wall in 10% and both anterior and posterior wall in 61% (Wallis and Donald 1988). In about 9% of the anterior wall fractures and 8% of the isolated posterior wall fractures, dural injuries occur; whereas in about 45% of the combined anterior and posterior wall fractures, dural lacerations are diagnosed (Wallis and Donald 1988).

Combined fractures of the posterior frontal sinus wall and the ethmoid (type III) occur in about 32% of craniofacial traumas. The combination of fractures of the ethmoid and sphenoid, respectively the ethmoid, cribriform plate and sphenoid (type II), is seen in 30.5%. In 25% of the cases there is a combination of anterior sinus wall, respectively anterior sinus wall, orbital roof and sphenoid fractures (type I) (Schroeder 1993).

Frequency of fractures/combined fractures in the region of the frontal sinus and skull base (Schroeder 1993)

Type I: Anterior sinus wall or anterior sinus wall roof of the orbit sphenoid fracture	25%
Type II: Ethmoid sphenoid or ethmoid cribriform plate sphenoid fracture	30.5%
Type III: Posterior sinus wall ethmoid fracture	32%
Type IV: Posterior sinus wall ethmoid sphenoid fracture	12.5%

Regarding the relative risk of concomitant dural injuries, posterior sinus wall fractures (fracture index*: 0.37) bear a higher relative risk than ethmoidal roof fractures (fracture index*: 0.17). Injuries at the transition from the posterior sinus wall to the ethmoidal roof (fracture index*: 0.15), respectively the roof of the orbit (fracture index*: 0.09), bear a lower risk Godbersen and Kgelgen (1998a).
(Fracture index*: Dura injury/fracture localization (<1.0))

5.4.1 Frontal Sinus: Midface Fractures

Frontal sinus fractures frequently coincide with orbital fractures and midface fractures (Schneider and Richter 1993). The combination with midface fractures in the orbital region is seen in 46% of the cases. In 34% the nasal bone is involved and in 12% the zygomatic bone.

On average there is an additional midface fracture on lower levels than the orbit in 15% of the cases Godbersen and Kügelgen (1998b).

Average involvement of the facial skeleton in frontal sinus wall fractures (Godbersen and Kügelgen 1998a, b)

Orbit	46%
Nasal bone	34%
Maxilla	15%
Zygomatic bone	12%

The involvement of the facial skeleton in frontal sinus fractures increases depending on the severity of the traumatic impact. In 53% of the anterior frontal sinus wall fractures (type I), additional fractures of the midface are found (12% of them have classic midface fractures). In fractures of the posterior frontal sinus wall (type II or type III), 95% additional fractures of the midface are found (25%, respectively 23% of them have classic midface fractures) Godbersen and Kügelgen (1998b).

Frequency of additional midface fractures in relation to craniofrontal fracture types (Godbersen and Kügelgen 1998a, b)

Craniofrontal type of fracture	Additional midface fracture
Anterior sinus wall fracture (type I)	12%
Posterior sinus wall fracture (type II)	25%
Posterior sinus wall fracture with involvement of the dura (type III)	23%

There was a clear peak in our own patients of between 20 and 50 years of age. All together, 62% of the patients were between 16- and 45-years old when the accident occurred (Neidhardt 2002). The group of 16–30 years was the largest, with 38% of all craniofacial fractures. Comprising 24%, the group of 31–45 years was the next largest, followed by the group of 46–60 years with 17%. Also significant were the 10% that were children between 1 and 15 years of age and the 11% of patients between 60 and 90 years.

The average age of a patient at the time of accident was 35 years. Approximately 80% of all craniofacial traumas fall in the category of the active working population between 16-and 60-years of age.

Distribution of craniofacial fractures according to age (Neidhardt 2002)

Age	Percentage (%)
1–15	10
16–30	38
31–45	24
46–60	17
61–75	7
76–90	4

Between 10 and 15% of the casualties with craniofacial fractures were children between 1- and 15-years old. Established reasons for craniofacial fractures in children are falls from a dresser or high bed (50%) as well as traffic accidents (50%) (Probst et al. 1990; Tarantino et al. 1999) (Fig. 5.2).

5.5 Distribution According to Age

Most of the patients suffering from craniofacial and skull base fractures are between 20 and 40 years of age (Probst 1971; Hill et al. 1984; Probst 1986; Weerda 1995).

5.6 Distribution According to Gender

According to Godbersen and Kügelgen (1998a, b) men are significantly more involved in craniofacial and frontobasal trauma than women. Comparable research shows

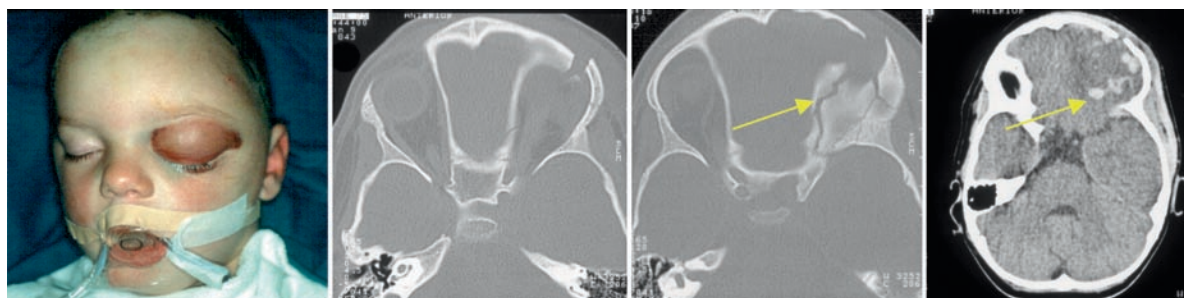


Fig. 5.2 Fracture of the frontal skull base and orbital roof (arrow) with hemorrhagic contusions in the left frontal lobe (arrow) in an 11-month-old child

the same results [Hill et al. 1984 (83%); Probst and Tomaschett 1990; Neidhardt 2002 (92%)]. Within our own trauma victims, the ratio of men to women was 11: 1 (Neidhardt 2002).

5.7 Associated Injuries

Of the patients with facial trauma, 50–70% show associated injuries (Gwyn 1970; Dufresne et al. 1992; Serletti and Manson 1992; Lehmann et al. 2001).

5.7.1 Thoracic, Abdominal, and Cervical Spine Injuries

Complicated polytrauma occurs in approximately 25% of all panfacial fractures. Nine percent of the cases concern abdominal and thoracic trauma (Smith and Bradley 1986; Schilli and Joss 1991; Shockley 1993). Twenty percent of the polytraumatic cases have additional injuries of the extremities (Joos et al. 2001). Approximately 10% have additional fractures in the cervical spine (Fig. 5.3).

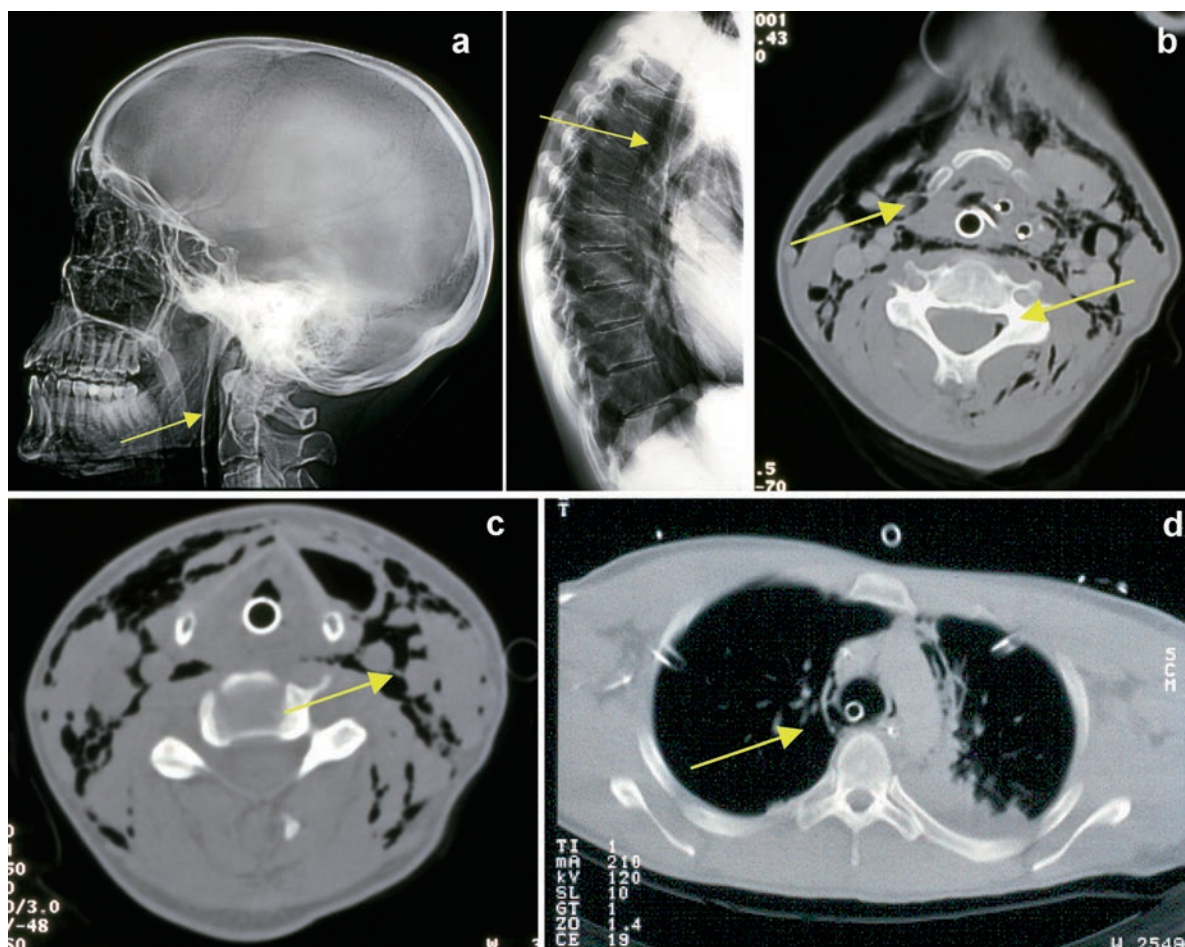


Fig. 5.3 Extended cervico-thoracic emphysema after tearing of the trachea in a mandibular and midface fracture. (a) Lateral cephalogram: comminuted and displaced fracture of the mandible and prevertebral collection of air (arrow). (b) CT scan:

emphysema spreading through the neck and into the spinal canal (arrow). (c) CT scan: cervical emphysema and asymmetry of thyroid cartilage and air collection within the parapharyngeal soft tissues (arrow). (d) CT scan: Mediastinal air collection (arrow)

- In craniofacial trauma, a timely control and diagnosis of additional injuries is essential

Extra attention should be paid to additional compression fractures and to luxations or fractures of the spine, particularly the cervical spine in unconscious patients or in patients with initial neurological symptoms. Surgical treatment has priority and the polytrauma protocol is followed (Potthoff 1985; Schweiberer et al. 1987; Ruchholtz et al. 1997; Piek and Jantzen 2000; Kuttenger et al. 2004).

5.7.2 Eye Injuries

Nearly 20% of the craniofacial trauma patients have serious eye injuries (Ioannides et al. 1988). These are mainly cornea-eyelid injuries, perforated eyeballs and injuries of the canthal ligaments (Neubauer 1987; Hardt 1989; Brandes et al. 1997; Brown et al. 1999; Rohrbach et al. 2000).

Complex periorbital trauma should be approached systematically by an ophthalmologist. The ocular and periocular traumas listed beneath are a suggested order of priority in addressing orbital and periorbital injuries (Fig. 5.4):

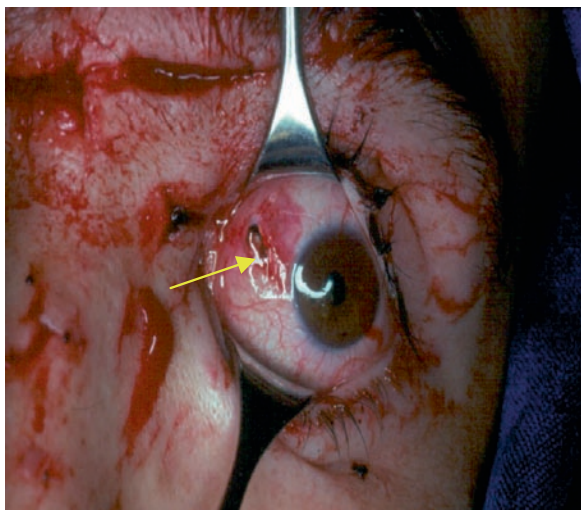


Fig. 5.4 Perforating injury of the orbital globe by a glass splinter (arrow). Periorbital soft-tissue laceration

- Cornea, globe, optic nerve, ocular muscles
- Lacrimal drainage system
- Medial canthal tendon
- Lid margins
- Lateral canthal tendon
- Levator muscle and aponeurosis
- Penetrating trauma of the eyelids and periocular region

5.7.3 Facial Soft-Tissue Injuries

Midface fractures often involve the facial soft tissues due to the traumatic impact. Contusions, skin abrasions, lacerations, tissue avulsions or burns and extensive and deep penetrating injuries are commonly seen in craniofacial injuries (44%) (Joos et al. 2001; Eppeley and Bhuller 2003) (Figs. 5.5 and 5.6).

5.8 Special Fractures and Complications

5.8.1 Penetrating Injuries

A very special pattern of craniofacial injuries is related to spin-off-fragments of various sizes while milling or sawing different materials. These fragments are loaded with high energy and can penetrate through the eye or demolish facial structures and penetrate intracranially (Figs. 5.7 and 5.8).

5.8.2 Gunshot Wounds and Tissue Avulsion

Gunshot wounds and tissue avulsions can lead to disastrous wounds due to soft- and hard-tissue defects. After primary wound closure, a plan for defect reconstruction has to be set up. Bone and soft-tissue transplants may be necessary to reconstruct the anatomy as far as possible.

A functional prosthetic rehabilitation based on implants is as important as epitheses in reconstructing

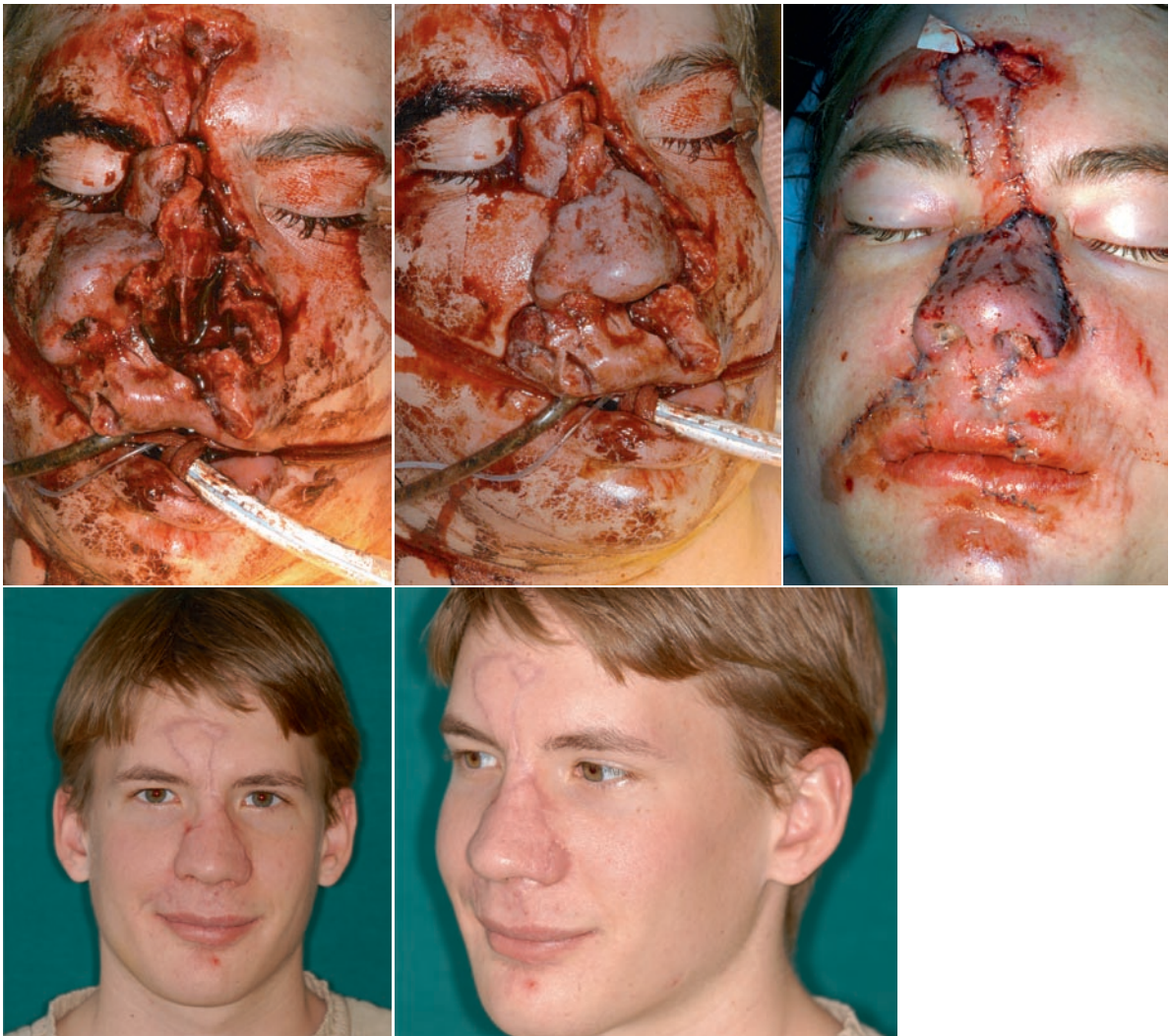


Fig. 5.5 Extensive soft-tissue laceration in the midface with subtotal amputation of the nose and naso-maxillary fracture (pre-postoperative)

Fig. 5.6 Severe injury to the central midface with soft-tissue laceration and complex naso-orbito-maxillary fracture (caused by a fraise). Preoperative situation and final result after reconstruction. There is residual ptosis of the left eyelid caused by nerve damage

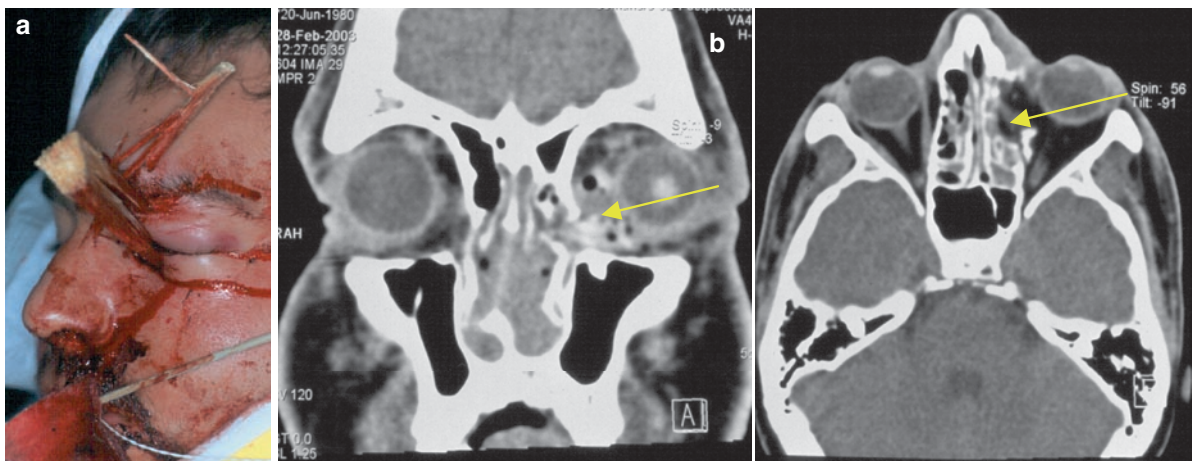


Fig. 5.7 (a) Perforating subcranial medio-orbital injury caused by a piece of wood. (b) Coronal and transverse CT images demonstrating the wooden splinter and substantial hematoma in the

inferomedial quadrant of the orbit with perforation of the nasoethmoidal wall (*arrow*). The globe is displaced laterally

Fig. 5.8 (a) CT image: perforating foreign body (wooden knot) injury from the left naso-orbital groove across the right orbit (arrow) with transection of the optic nerve. (b) After binasal exploration and removal of the foreign body: amaurosis, ophthalmoplegia, and exophthalmus

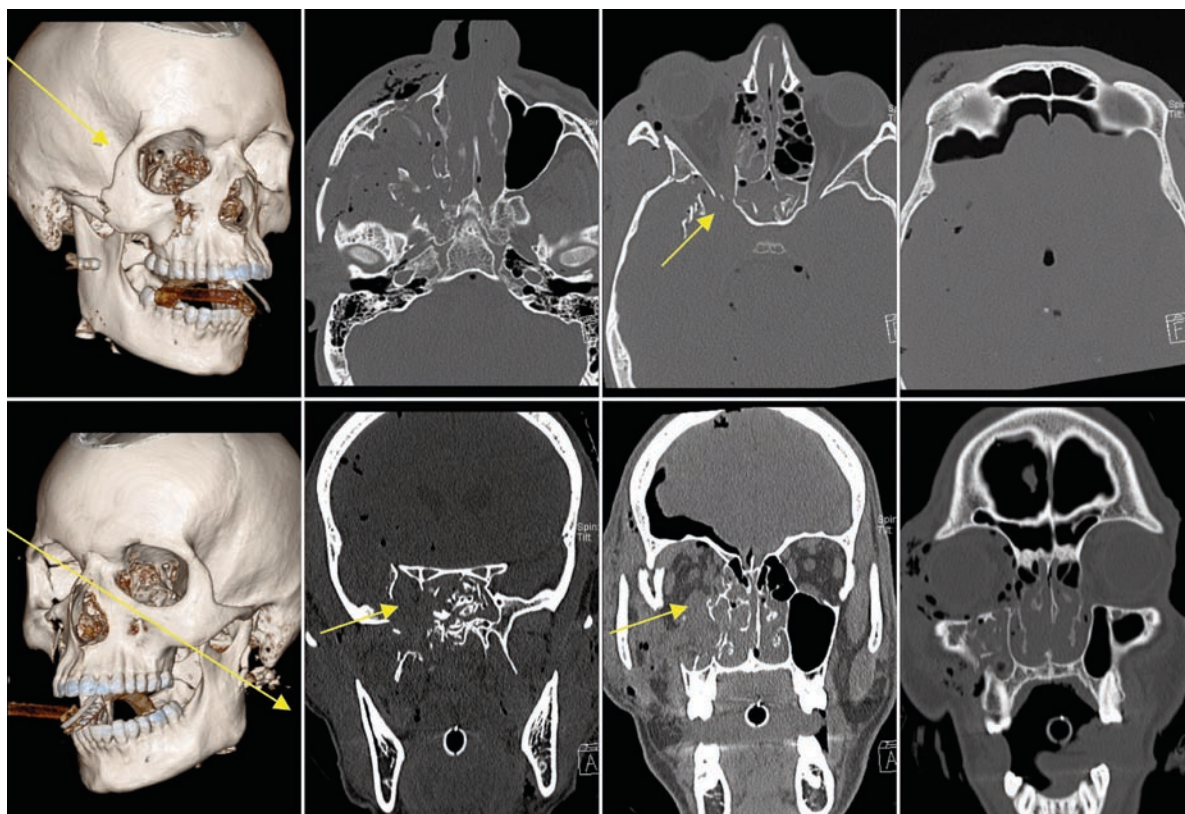
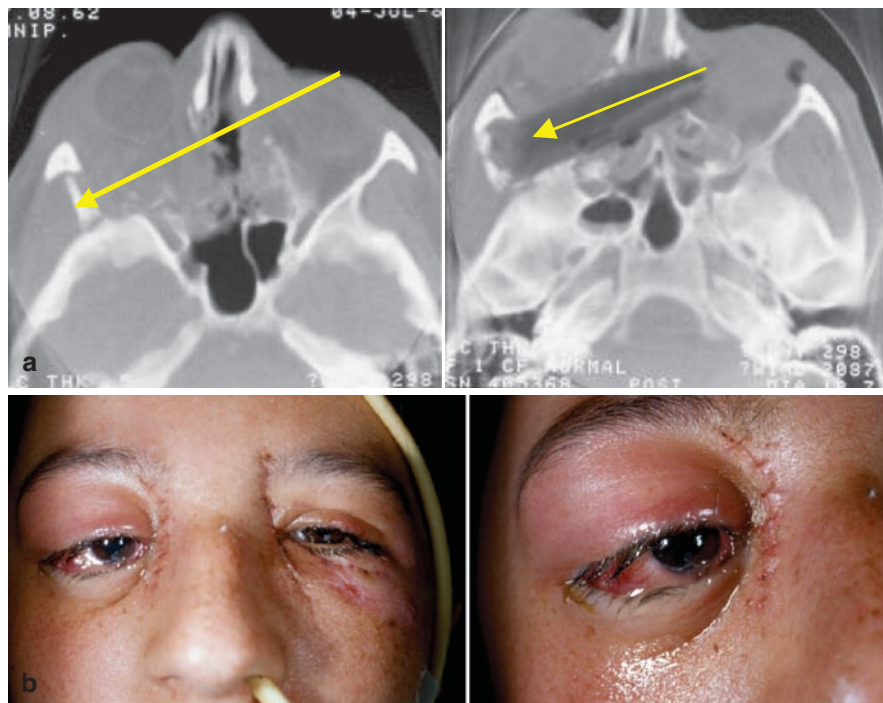


Fig. 5.9 Craniofacial gunshot wound. Destruction of the lateral midface and the skull base in the ethmoido-sphenoidal complex (arrows)

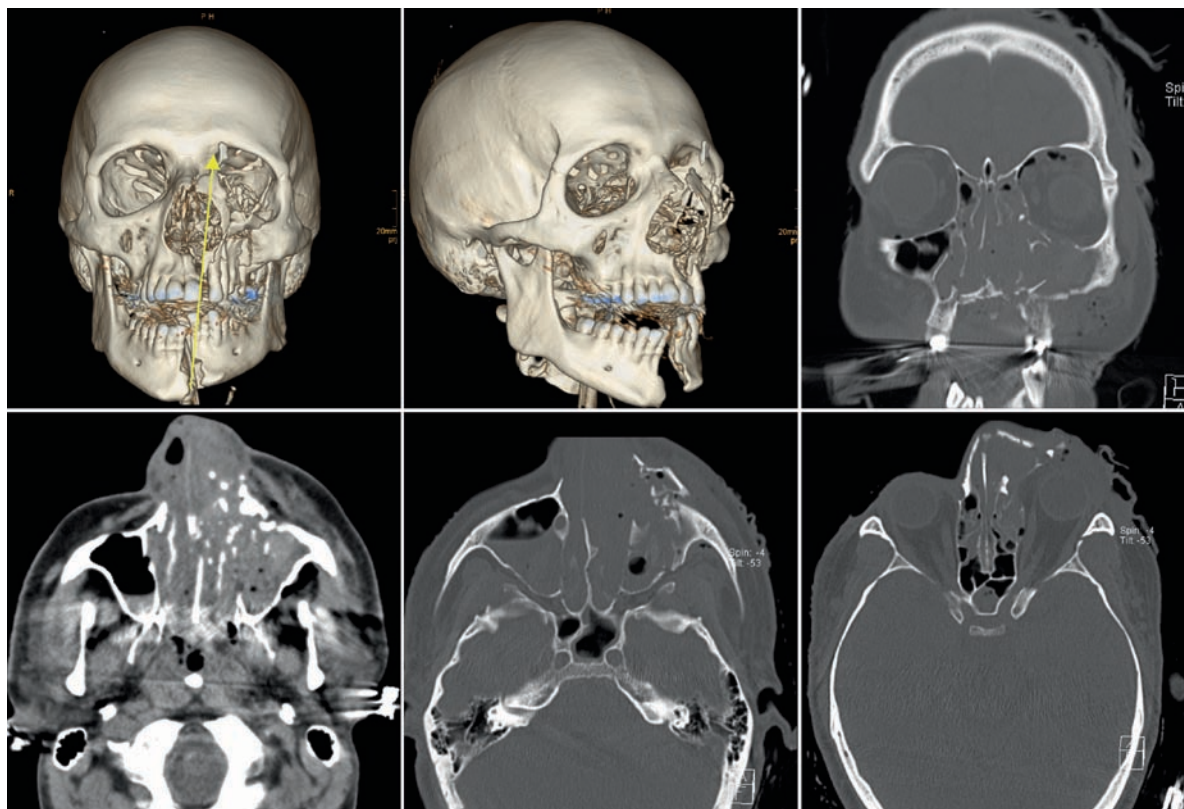


Fig. 5.10 Subcranial burst fracture of the mandible and maxilla after gun shot trauma from submental into left maxilla and the naso-orbito-ethmoidal region (arrow)

defects for aesthetic reasons. Satisfying results are not always possible (Figs. 5.9–5.11).

5.8.3 Complicating Effects

Complicating effects of fractures in the craniofacial complex can lead to essential loss of function.

5.8.3.1 Nose: Nasal Septum – Nasolacrimal Duct

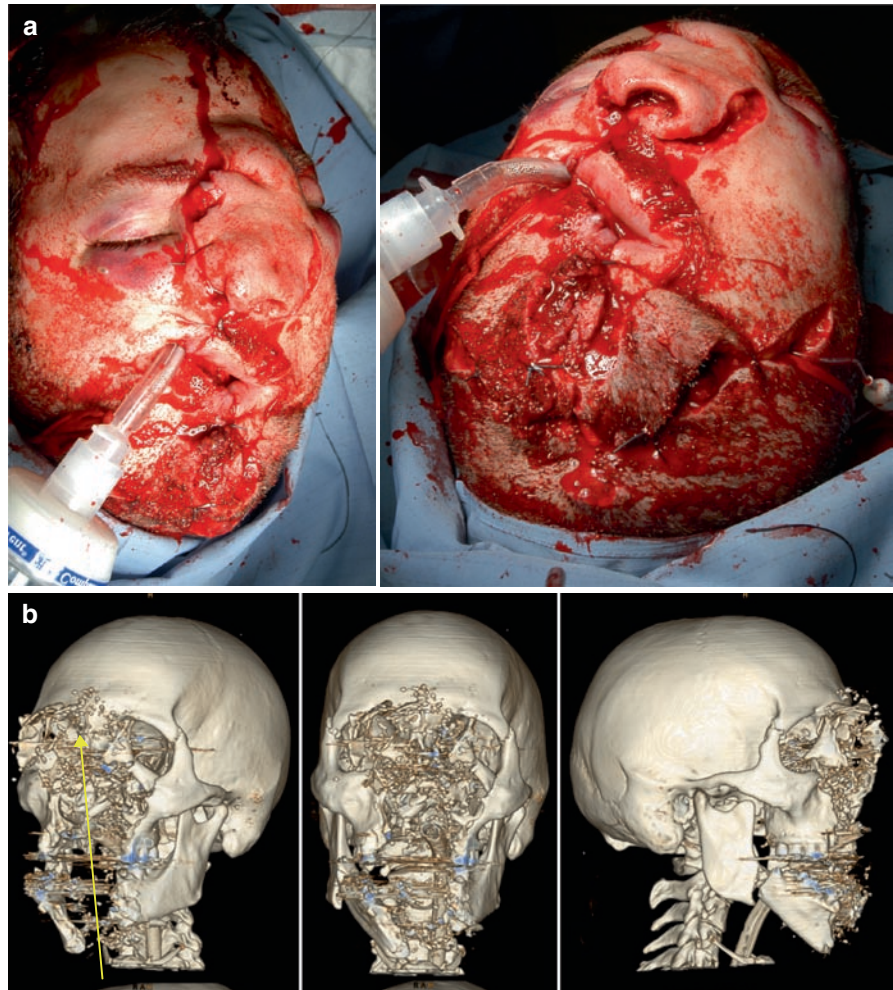
Every fracture in the midfacial region can lead to an obstruction of the respiratory pathways and a functional interference with nasal respiration. Traumatic destruction of the paranasal sinus system can result in chronic infections with loss of gustatory function, formation of cysts and atypical neuralgiform facial pain attacks. Fractures and dislocations of the nasal septum and the nasal bone can lead to obstruction of the nasal pathway. Chronic

infections in the paranasal cavities may result from traumatic obliteration of the natural nasal pathways (Mathog et al. 1995; Theissing 1996) with disruption and obstruction of the nasolacrimal duct.

5.8.3.2 Orbit

Midface fractures frequently lead to fractures of the orbital walls. Prevalently the orbital floor and the medial walls are affected. Fracture lines running through the posterior orbital apex endanger the optical nerve. A permanent loss of vision might be the consequence. A traumatic dislocation of orbital tissues into the maxillary sinus is frequently seen. Enophthalmus and hypophthalmus are typical clinical consequences. Dislocation of the globe and a mechanical blockade of the periorbital muscles will lead to double-vision and disturbances of eye motility. Even a traumatic dislocation of the eye into the maxillary sinus is possible.

Fig. 5.11 Burst trauma of the midface and the mandible after severe suicidal gun shot trauma from submental through midface. **(a)** Clinical situation after emergency intubation. **(b)** CT: extensive destruction of the central midface and loss of the lower central midface structures (*arrow*) including the palate



CMF may also lead to tearing off the medial or – less often – the lateral canthal ligaments. Reconstructing the osseous orbital walls and repositioning the canthal ligament insertion is of utmost importance in the primary surgical intervention. A secondary reconstruction will lead to less satisfying results (Rowe and Williams 1985; Dutton and Al Qurainy 1991; Mathog 1992; Mathog et al. 1995; Rohrbach et al. 2000).

5.8.3.3 Ethmoid

Midface traumas may be combined with skull base fractures. The cribriform plate of the ethmoid bone is most frequently affected, creating a penetrating defect between the neuro- and viscerocranium. Liquor fistulas, ascending infections, persisting olfactory disturbance

and traumatic damages to the brain are possible consequences (Theissing 1996; Ernst et al. 2004).

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6.1 Combined Skull Base and Midface Fractures

Apart from the classical midface fracture signs, there are certain (direct) and uncertain (indirect) clinical symptoms indicating additional skull base fractures. Liquorrhea, clinically visible bony prolaps, dislocated bony fragments and intracranial air (CCT) or dislocated skull base fragments (CCT) indicate skull base fractures with dural laceration (Hausamen and Schmelzeisen 1996; Messerklinger and Naumann 1995; Joss et al. 2001).

Signs of open skull base fractures

- Clinical evidence of a liquorrhea
- Brain prolapse
- Visible bone defects due to dislocated bony fragments
- Intracranial air in CCT
- Dislocation of skull base fragments, basal fissures and bone diastasis in CCT

6.1.1 Certain Signs of Skull Base and Dural Injuries

6.1.1.1 Liquorrhea

Nasal liquorrhea is an obvious sign of an open skull base fracture (Probst 1971, 1986; Entzian 1981; Loew et al. 1984; Weerda 1995). Nasal liquorrhea indicates a liquor fistula, an open communication between the intracranial intradural space and the pneumatized sinuses of the viscerocranium.

Liquor loss from the left or the right nostril might indicate the location of the osseous skull base defect, although this cannot be relied on. The sinus septum can also be damaged in fractures of the posterior wall of the frontal sinus, thus causing diagnostic confusion as liquor might drain on the opposite side. On the other hand, liquorrhea from both nostrils does not necessarily indicate a bilateral cranio-nasal fistula (Kessel et al. 1971; Kastenbauer and Tardy 1995).

Fistulas

There are varying excretory pathways for liquor (Loew et al. 1984; Rosahl et al. 1996). The liquor can flow directly through an osseous defect in the cribriform region. Indirect pathways run through fractures in the paranasal sinus system using natural drainage outlets: e.g., the nasofrontal duct, via the ethmoid bone or over the drainage system of the sphenoid sinus.

According to Waller (1977), direct cranio-nasal fistulas (cribriform plate fistulas) are the cause of liquorrhea in 51% of the cases, whereas indirect cranio-sino-nasal fistulas (frontonasal/fronto-ethmoidal-nasal/sphenoidal-nasal) represent 48% of the cases. Sphenoid fistulas occur in about 5% of the cases (Fig. 6.1).

Multiplicity

According to Probst and Tomaschett (1990), 59% of the frontal skull base injuries are associated with dural injuries. Thirty-five percent of the dural tears are unilateral, 24% bilateral and in 44% of these patients multiple dural lacerations are found.

In unilateral fractures with dural lacerations, 79% of the dural tears are ipsilateral and 21 % bilateral, in

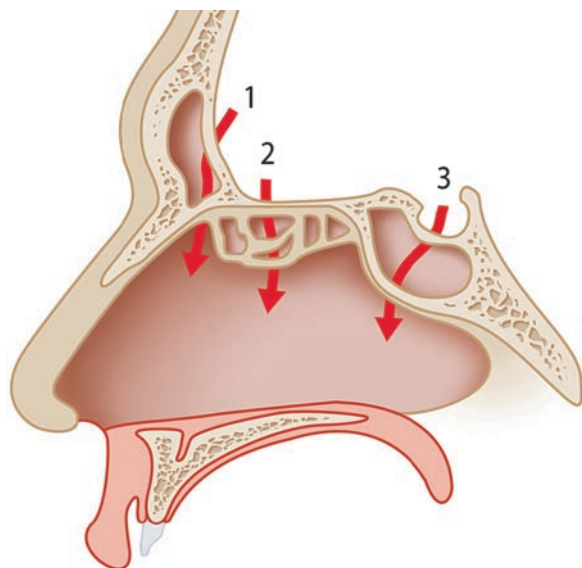


Fig. 6.1 CSF leaks: the most frequent locations of dural defects after frontal skull base injuries (mod. a. Ewers et al. 1995). 1 Fronto-nasal CSF leak (direct cranio-nasal), 2 fronto-ethmoido-nasal CSF leak (indirect cranio-sino-nasal), 3 sphenoidal CSF leak (indirect cranio-sino-nasal)

bilateral fractures 65% of the dural lacerations are bilateral and 33% unilateral located (Schroth et al. 2004).

According to Lewin (1974), bilateral dural tears occur mostly in central midface injuries (80% bilateral dural fistulae). According to Godbersen and Kügelgen (1998), one can expect several dural tears, especially in frontocranial injuries of type III (posterior sinus wall fractures).

In lateral skull base fractures, however, only 20% of the patients have dural injuries, but in 52% multiple dural lacerations are found (Lewin 1974).

In our own patients, approximately 56% of the craniofacial fractures had concomitant dural injuries, of which 12% were bilateral and 44% unilateral.

Time of Manifestation

There is a distinction between early and late onset of liquorrhea (Lewin 1974; Spetzler and Zambranski 1986; Kaestner et al. 1998).

Statistically, a liquorrhea starts in 63% of the cases within the first 24 h following trauma. In 51–80% of the cases the onset of a liquorrhea can be observed within 48 h (Lewin 1974; Fournier 2007). A late onset between 2 and 12 weeks following the craniofacial

injury occurs in 30% of the cases, in 14% after 2–6 months and in 7% after 7–12 months (Lecuire and Mounier-Kuhn 1961).

Average manifestation of liquorrhea following frontal skull base injury, in relation to posttraumatic time (Lewin 1974)

0–48 h	51%
1–7 days	8%
2–4 weeks	18%
2–3 months	12%
4–6 months	2%
6–12 months	2%
>1 year	7%

The eventuality of a late onset of liquorrhea does not exclude a dural injury in skull base injuries (Paillas et al. 1967). However, one has to keep in mind that a clinically apparent liquorrhea may stop spontaneously within a week in about 85% of the patients (Georgiade et al. 1987; Schmidek and Sweet 1988) (see also Sect. 7.2)

A clinically apparent loss of liquor weeks or even months following trauma may be caused by necrosis of brain tissue squeezed between fractured bones. The necrosis destroys the dural barrier. As the arachnoidal space is only separated from the paranasal sinuses by incomplete and unstable scarred connective tissue (Russell and Cummings 1984; Kaestner et al. 1998), a sudden intracranial pressure increase may cause the scar tissue to break and a liquor fistula will appear (Probst 1986).

Clinical Evidence of Liquorrhea

Every suspicion of liquorrhea must either be confirmed or excluded by further examination. Standard clinical methods are:

- **Rhinoscopy:** obvious liquor loss from the paranasal sinuses, visualization of defects
- The “handkerchief” test: nose secretion does stiffen on a handkerchief, liquor does not
- The swab/sponge test: liquorrhea gives a liquor border around the bloodstain, whereas regular nose secretion does not
- The Queckenstedt test: pressing the jugular vein will raise the intracranial pressure resulting in increased liquor leakage
- Nasal endoscopy: with the aid of fluorescence identification

Chemical Liquor Diagnostic

Glucose-Protein Test

Liquor has a higher glucose and protein concentration than nasal secretion; hence, by determining the concentration of glucose and protein in a fluid, a differentiation can be made between regular nasal secretions and liquorrhea.

A laboratory-tested glucose concentration >40 mg% and a protein level <100 mg% (maximum 200 mg%) characterizes liquor. However, liquor testing for glucose and proteins with test sticks is not safe.

Immunological Liquor Diagnostic

Beta-2 Transferrin Determination

Nowadays the immunological evidence of beta-2 transferrin in a liquid suspected to be liquor is the standard test (Oberascher and Arrer 1986; Ernst et al. 2004). It should always be performed to exclude a dural defect with a liquor fistula. If there is a suspicion of liquorrhea, even with a negative test result (3% of the results are false negative), the beta-2 transferrin test method can be improved by marking the liquor (see below) (Bachmann et al. 2004).

Beta-Trace Protein

Beta-trace protein, which has a higher specificity than beta-2 transferrin, is preferentially used to diagnose liquorrhea (Ernst et al. 2004; Bachmann et al. 2004).

Liquor Marking Methods

The marking test agent (sodium fluoride/Na-fluorescein) has to be instilled intrathecally by lumbar puncture. Later the marked fluid leaking through open defects in the nose or the sinuses may be detected by nasal endoscopy.

Additionally high-resolution *coronal computed tomography (CT)* of the paranasal sinuses and the anterior skull base, a *Jotrolan CT* or an *MRI* and, if necessary, a *liquor scintigraphy* (In111/Tc 99-DTPA) may be integrated to detect dural defects with liquor leakages (Goshujra 1980; Chow et al. 1989; Horch et al. 1991). These methods should be applied in situations of unclear or recurrent liquor loss and are apt not only to detect small dural leakages but also help to localize the defect site (Ernst et al. 2004).

The combined use of different liquor leakage detecting methods guarantees an exact diagnosis and results in better preoperative planning (Ernst et al. 2004) (Fig. 6.2).

Concept of detecting liquor fistulas (Ernst et al. 2004)

Depending on the individual situation, diagnosis of liquor fistulas can be based on the following types of tests:

- Clinical
- Chemical (glucose-protein)
- Immunological (beta-2 transferrin/beta-trace protein)
- Liquor marking methods (endoscopic Na-fluorescein identification/liquor-scintigram)
- High-resolution CT/CT-based cisternography

Fig. 6.2 Submucous CSF collections bilateral to the nasal septum (arrow) in a patient with frontal skull base and subcranial mid face fracture



6.1.1.2 Pneumocephalus

An important symptom of skull base and dural injuries is the intracranial accumulation of air (pneumatocele, pneumocephalus, pneumo-encephalocele). An accumulation of air can be found in the subdural, subarachnoidal, intracerebral, or intraventricular space. Various combinations are possible.

A primary pneumatocele develops immediately after trauma. Air accumulates in the subdural or subarachnoidal space. A secondary pneumatocele results from a liquor fistula. Air enters the arachnoidal space via the liquor fistula and spreads from there to the basal

cisternae and into the ventricular system. In rare cases, the air is even found in the third ventricle.

If a dural tear conglutinates secondarily, a unidirectional valve mechanism can result: with increasing intracerebral pressure, e.g., while coughing or sneezing, air enters the cerebral tissue via the fistula and leads to an enlargement of the pneumatocele.

An epidural pneumatocele mostly occurs unilaterally, is less extensive than the subdural pneumatocele and remains stable even when changing the patient's position.

Subdural pneumatoceles often extend over the whole surface of the cerebral hemisphere and often also along the falx cerebri or over the tentorium

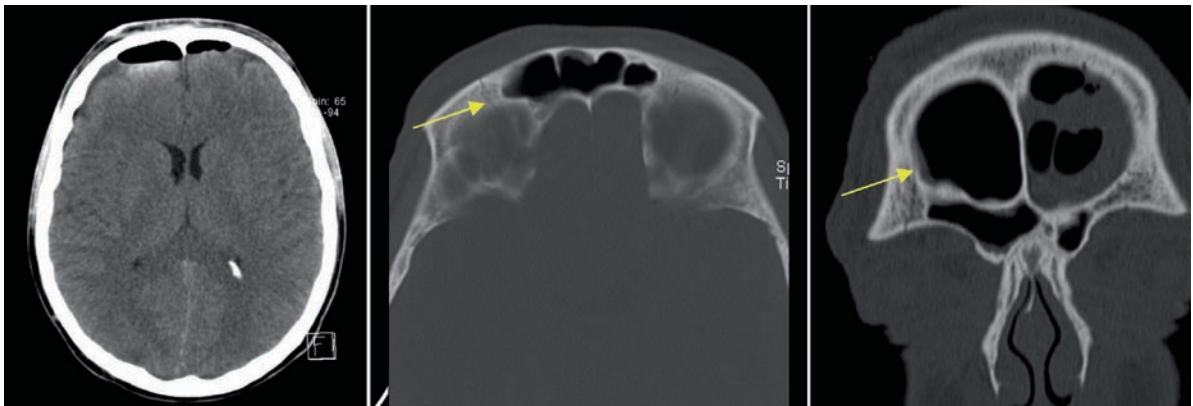


Fig. 6.3 Small fissural fracture in the frontal bone with associated injury to the dural membrane. Consecutive large frontal pneumatocele, indicating an open skull fracture. The fissural fracture line crosses the lateral margin of the frontal sinus (arrow)

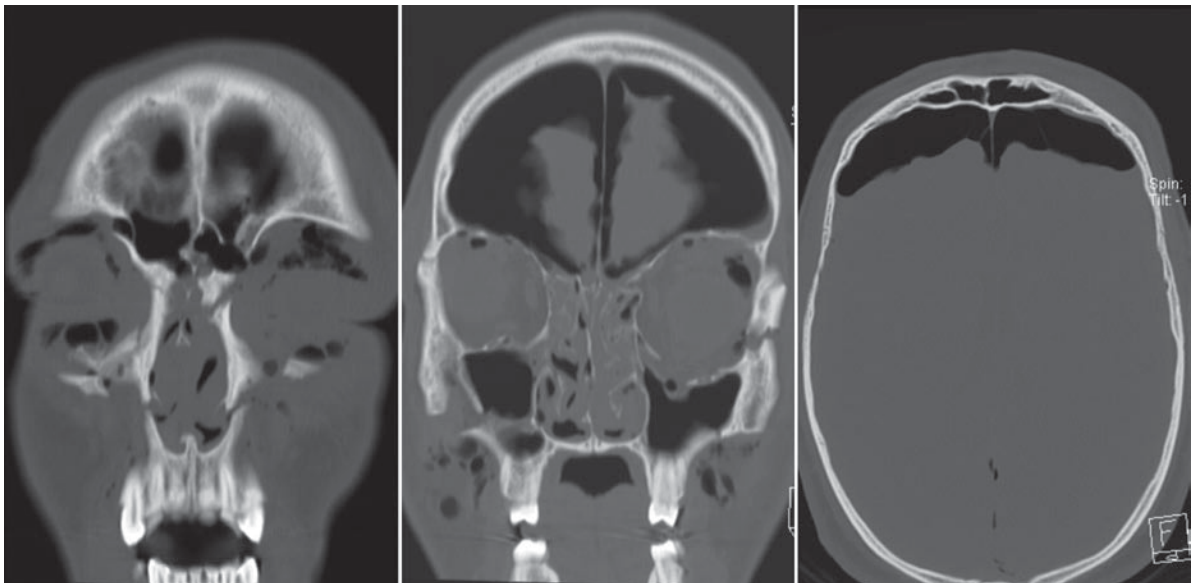


Fig. 6.4 Pneumocephalus associated with a complex comminuted fracture of the midface and frontal skull base (CCMF fracture)

cerebelli and change their extension whilst changing the patient's position.

Subarachnoidal and intraventricular pneumatoceles impose on X-rays as a pneumo-encephalogram.

Pneumatoceles are a permanent risk of infection and will lead to rising intracranial pressure, especially if there is a unidirectional valve mechanism (Kretschmer 1978).

According to Probst and Tomaschett (1990), a major pneumocranium occurs in 22% of the cases with skull base fractures. In our own patient group with craniofacial fractures, 27% of the patients developed a pneumocranium (Figs. 6.3 and 6.4).

- It is often not possible to detect liquor leakages in the acute phase of craniofacial injuries. The radiological evidence of a pneumatocephalus is regarded as evidence of a possible dural injury

6.1.1.3 Meningitis

If therapy and trauma management are inadequate in open *skull base traumas*, the direct communication of contaminated, bacteria-loaded tissues or spaces within the intracranial space results in a potentially lethal infection risk (Entzian 1981; Dagi and Poletti 1983; Süss and Corradini 1984; Wilson et al. 1988; Wolfe and Johnson 1988; Hell et al. 1996).

The start of meningitis from within a few hours up until the first days following the accident is known as early meningitis. To ensure the diagnosis, a liquor puncture should be performed (Potthoff 1985).

The potential risk of developing meningitis in frontal skull base fractures varies from 25% (Jamieson and Yelland 1973; Flanagan et al. 1980; Dagi et al. 1983; Hubbard et al. 1985; Schmidek and Sweet 1988) to over 30% (Eljamel and Foy 1990), with a cumulative long-term risk of 85% within 10 years after the trauma. Surgical wound repair lowers the average risk from 30 to 4% and the cumulative risk from 85 to 7% (Eljamel and Foy 1990).

- The potential risk of a cerebral infection (meningitis, intracerebral abscess) lies between 25 and 30% in nontreated, traumatic frontal skull base defects (Loew et al. 1984; O Brian and Reade 1984).

The average risk of meningitis in a fracture of the posterior wall of the frontal sinus is calculated at 2.6–9%

(Hager 1986; Wallis and Donald 1988; Wilson et al. 1988; Miyazaki et al. 1991(1–3%); Schroeder 1993; Godbersen and Kügelgen 1998).

6.1.2 Uncertain Signs of Skull Base and Dural Injuries

6.1.2.1 Lesions of the Cranial Nerves

Various trauma mechanisms can lead to specific cranial nerve lesions. If such lesions are detected, there is a high probability of a skull base injury (Potthoff 1985).

In midface/skull base fractures, neurological complications of extracranial origin occur in about 50% of the cases (Bonkowsky et al. 1989). According to Lee (1983), consequences involving neurological ocular motility occur in 17% as lesions of the cranial nerves III–IV–VI (30% cranial nerve III/14.5% cranial nerve IV/34% cranial nerve VI and 21.5% combinations of these nerves).

Olfactory Nerves

Frontal skull traumas are the cause of most uni- or bilateral injuries to the olfactory nerves (I) (unilateral or bilateral anosmia)

Possible causes are:

- a direct injury to the olfactory fibers
- a disruption of the fibers in a fracture of the cribriform plate
- a trauma-associated temporary injury of the olfactory bulb or tract
- a destruction or irreparable contusion of the olfactory bulb
- a cortical contusion and a contre-coupe damage at the bottom of the frontal lobe of the brain.

Indirect injuries of the olfactory filaments occur by overextension of the fine filaments induced by contusional dislocation of the brain, bleeding in the area of the olfactory bulb or by a hematoma in the perineurium of the olfactory ligaments. Contre-coup frontal lobe contusions may also cause an alteration in olfactory function. Posttraumatic dysosmia occurs in 5–9% of the cases (Lewin 1966).

In 50% of the traumatic dysosmia cases there is a complete bilateral anosmia, in 20% a unilateral anosmia and in the other cases a unilateral or bilateral hyposmia. A hyposmia usually has a good prognosis; whereas in an anosmia, the prognosis is bad (Kretschmer 1978).

There are different possibilities to test the olfactory sense: subjective olfactory sense test/simulation tests/semiquantitative determination of the olfactory sense/objective olfactometry.

Oculomotor Nerve

The oculomotor nerve (III) is mostly damaged within the orbit or in the area of the superior orbital fissure. Ptosis, widened unresponsive pupils and immobility of the globe are characteristic for a complete loss of function of the oculomotor nerve. Partial paralysis is mostly limited to a ptosis, a restriction of cranial eye motility and abnormal pupil reaction.

Three outcomes are possible: (1) no recovery, (2) regenerative recovery and (3) aberrant regeneration.

Recovery may take between 6 and 9 months. When aberrant regeneration occurs, paradoxical eye movements are observed. Typically, eyelid elevation occurs on attempted adduction or downward gaze. Pupil constriction and accommodation may accompany downward gaze, with pupil dilation on abduction (Dutton and Al-Qurainy 1991) (Fig. 6.5).

Trochlear Nerve

Isolated paralysis of the trochlear nerve (IV) seldomly occurs in skull base fractures. This is mostly combined with defects in function of the oculomotor nerve. Reasons for damage are: skull base fractures in the region of the petrous bone, fractures of the greater wing of the sphenoid and of the temporal bone along the petrosquamosal fissure. Also intracranial injuries located at the top of the pyramids of the petrous bone can damage the nerve (Kessel et al. 1971; Kretschmer 1978). Frontal antero-posterior trauma is the most common cause.

The superior oblique muscle, which is innervated by the trochlear nerve, is responsible for depression of the globe in adduction and also produces intorsion. Vertical diplopia, which may be accompanied by torsional double vision, occurs in case of damage to the trochlear nerve. Spontaneous recovery may occur 3–6 months after injury (Dutton and Al-Qurainy 1991).

Abducent Nerve

The abducent nerve (VI) has a long intracranial course and is frequently damaged by direct trauma. Paralysis mostly occurs after skull base fractures, especially in fractures in the area of the petrous bone — or as a consequence of raised intracranial pressure (ICP). Examination reveals a paralyzed lateral rectus



Fig. 6.5 Partial paresis of oculomotor nerve after right orbito-zygomatic fracture. The abducent nerve is still intact

muscle and failure of ipsilateral abduction. Abducent nerve palsy may also result from a traumatic carotico-cavernous sinus fistula.

Optic Nerve

The optic nerve is damaged in 2% of all closed traumatic cranial injuries (Holt and Holt 1983; Gossman et al. 1992) and in 20% of all frontal skull base injuries (Ioannides et al. 1988). The highest risk of damage exists in frontal (72%) and fronto-temporal (12%) traumas (Kline et al. 1984; Sofferman 1988, 1991).

Direct lesions of the optic nerve can be observed in skull base trauma caused by compression of the nerve during its intracanalicular course and/or by dislocated bone fragments compressing the nerve (Hardt and Steinhäuser 1979; Lädach et al. 1999).

Indirect lesions result from contusion, necrosis, rupture of the vessels, intracanalicular or/and intracranial hematomas. Also, secondary edemas and circulatory problems put the nerve function at risk. Indirect lesions are seen in 6.1% of the skull base fractures (Obenchain et al. 1973; Mathog 1992) (Fig. 6.6).

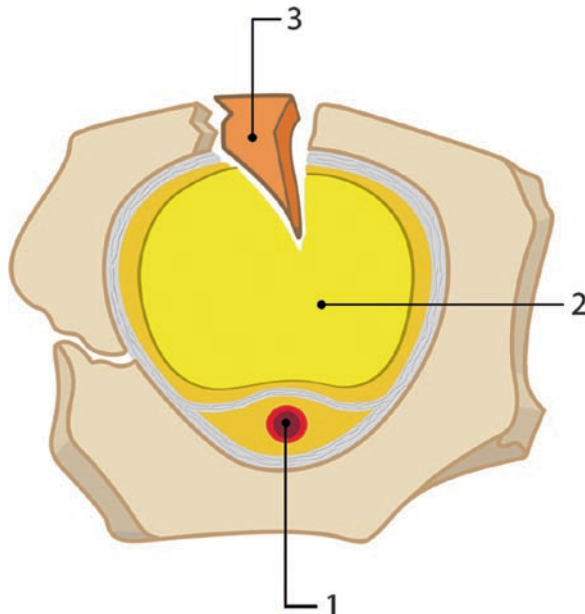


Fig. 6.6 Mechanism of injury to the optic nerve (*schematic drawing*). Indirect forces to the sphenoid bone lead to fragmentation of the sphenoid wing with intrusion/compression of fragments into the optic nerve (mod. a. Hardt and Steinhäuser 1979, Lädach et al. 1999). 1 Ophthalmic artery, 2 optic nerve, 3 bone fragment (sphenoid)

- A progressive loss of vision without injury to the globe or the bony orbital cone is most likely caused by an orbital hematoma and/or compression of the optic nerve in its intracanalicular pathway due to bleeding or edema (Lipkin et al. 1987; Stoll 1993; Rochels and Rudert 1995).

Loss of Vision in Midface Fractures

A loss of vision in midface fractures occurs in 15-20% of all severe midface injuries (Jabaley 1975; Holt and Holt 1983; Al-Qurainy IA et al. 1991 a, b, c; Dutton and Al-Qurainy 1991; Brown et al. 1999; Poon et al. 1999; Cook 2002; Manolidis et al. 2002; Soparkar 2005).

Injury to the optic nerve most frequently occurs in central fronto-naso-ethmoidal fractures due to fractures along the lesser wing of the sphenoid and seldomly in complex lateral midface fractures with fractures of the greater wing of the sphenoid (Ketchum et al. 1976; Kretschmer 1978; Hardt and Steinhäuser 1979; Chilla 1981; Bleeker and Los 1982; Lipkin et al. 1987; Fonseca and Walker 1991; Vitte et al. 1993; Lädach et al. 1999; Soparkar 2005; Stewart 2005).

Nearly 76% of isolated fractures of the sphenoid bone are associated with lesions of the orbit. There is a deterioration of vision in about 29% due to direct or indirect injury to the optical nerve (Hasso et al. 1979; Ghobrial et al. 1986) (Figs. 6.7 and 6.8).

Location of Optic Nerve Lesions

The most susceptible region for injuries of the optic nerve is located in the intracanalicular pathway from the orbital cone to the optic chiasm (Ramsay 1979; Gellrich et al. 1997). In 56% of the cases this is the location of trauma (Kessel et al. 1971). There are almost always associated skull base fractures, yet in approximately only 10% of the cases the fracture lines run through the optic canal or through the anterior clinoid process (Ramsay 1979; Leider and Mathog 1995).

The second largest group of injuries comprises injuries located in the area between the optical papilla and the point where the central retinal artery enters the optic nerve. These make up 13% of the injuries.

Injuries resulting from nerve tearing at the junction point with the globe ball form the third largest group with approximately 11.6% (Kessel et al. 1971).

Fig. 6.7 Fracture of the sphenoid wing with compression of the optic nerve (*arrow*)

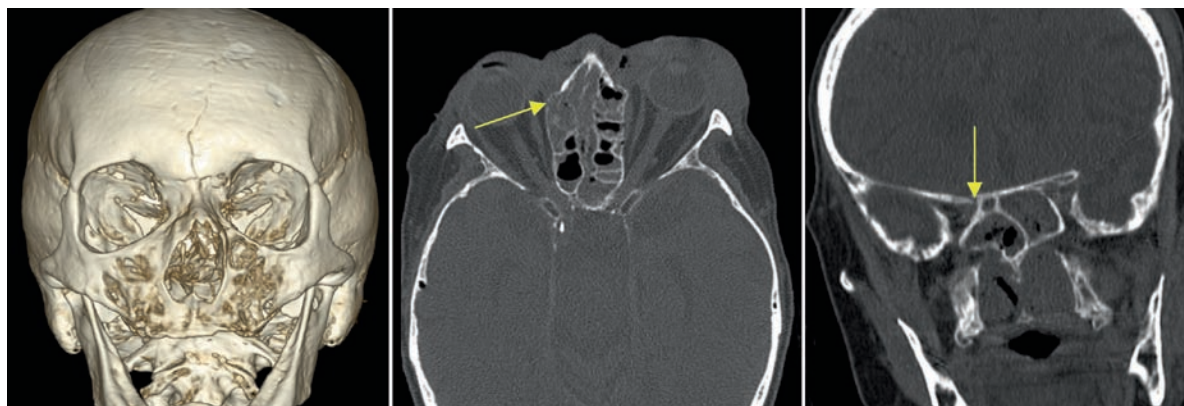
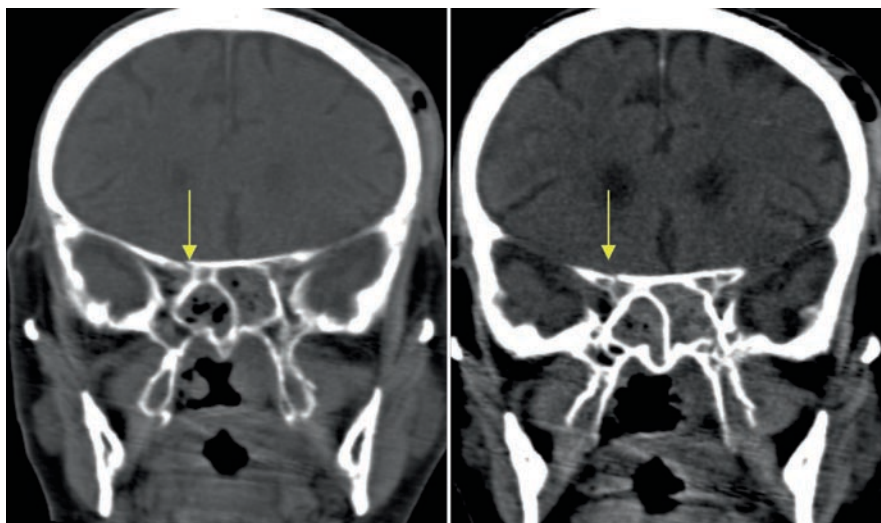


Fig. 6.8 Complex impression fracture of the midface with posterior dislocation of the ethmoid complex and fracture in the roof of the optic canal (*arrow*)

Clinical Appearance

Visual reduction may vary from reversible to irreversible loss of vision with the preservation of globe motility (Lipkin et al. 1987; Gellrich et al. 1996; Gellrich 1999). Clinically, either a bitemporal hemianopsia or an amaurosis (canalis opticus syndrome) with an intact concurrent reaction to light may develop in the involved eye.

In 34% of complex midface injuries with dysopia there is a reduction of vision, in 47–52% there is an immediate amaurotic damage and in 12–14% a protracted development of an amaurosis (Neubauer 1987) (Fig. 6.9).

CT Analysis of Optic Nerve Lesions (Gellrich 1999)

Primary CT Signs

- Fracture running through the optic canal, possibly with dislocated fragments
- Fracture in the retrobulbar orbital region, intraorbital bony fragments
- Soft tissue: optic nerve hematoma/edema, optic nerve swelling, interruption of the optic nerve, hemophthalmia
- Retrobulbar hematoma in the posterior third of the orbit

Fig. 6.9 Injury to the optic canal with amaurosis and internal ophthalmoplegia but undisturbed motility after a complex central midface fracture (CUMF fracture)



Secondary CT Signs

- Occlusion of the sphenoid sinus/posterior cells of the ethmoid sinus
- Epidural temporo-basal hematoma

Additional Injuries

- Fracture of the lamina papyracea/fracture of the frontal sinus
- Zygomatic fracture, fractures of the orbital floor/roof
- Air accumulation in the region of the optic chiasm, cavernous sinus, greater wing of the sphenoid
- Frontal – temporobasal contusions – subarachnoidal – subdural hemorrhage/hematoma

Operating Indications/Decompression

Decompression of the Orbital Cavity

The indication for decompression (orbitotomy) is given by an afferent disturbance of the optic nerve — amaurosis or progressive loss of vision — based on a retro bulbar hematoma. A liquor fistula, a pulsating exophthalmus (carotis-cavernous sinus fistula) and other general contraindications due to the trauma should be excluded.

Decompression of the Optic Canal

- Decompression of the optic nerve canal in conscious patient:

In the case of afferent nerve disturbances with progressive loss of vision or amaurosis based on radiological evidence of fractures in the retrobulbar orbital region or in the optic canal, surgical decompression of the nerve in the optic canal (transtethmoidal decompression of the optic nerve) should be performed as soon as possible.

- Decompression of the optic nerve in unconscious patient:

There is an indication for an operative transtethmoid decompression if there is direct or indirect radiological evidence of a retrobulbar trauma in the orbital region or in the neighboring region of the optic canal and if there is clinical evidence of an afferent disturbance of the optic nerve [alternative: presence of pathological visually evoked potentials (VEPs)] (Gellrich et al. 1996; Gellrich 1999):

Therapy/Prognosis

Decompression of the orbital contents and the optic canal is performed through an endonasal/transethmoidal, transfacial/transethmoidal, or a transfrontal/transcranial

neurosurgical approach. The operation has to be performed within the first 6 h following trauma (Yoshinai 1972; Fukado 1981; Krausen et al. 1981; Stoll et al. 1987, 1994; Sofferman 1988, 1991; Mann et al. 1991; Niho 1991; Mathog 1992; Rochels and Behrendt 1996; Koch and Lehnhardt 2000).

Decompression 8–10 h following trauma often results in a permanent reduction of vision or even an amaurosis (Kennerdell et al. 1976; Lädach et al. 1999).

Even following prompt decompression, both micro- and macro-traumatic injuries to the optic nerve with acute loss of vision only have a reduced chance of recovery. In less than 20% of the cases, normal or nearly normal vision returns (Beuthner 1974; Dutton and AL-Qurainy 1991; Rochels and Behrendt 1996, 1997).

If there is not an immediate but a slow reduction of vision (edema - hematoma in the optic canal), early decompression will be successful in 20–30% (Beuthner 1974; Osguthorpe and Sofferman 1988) (Figs. 6.10 and 6.11).

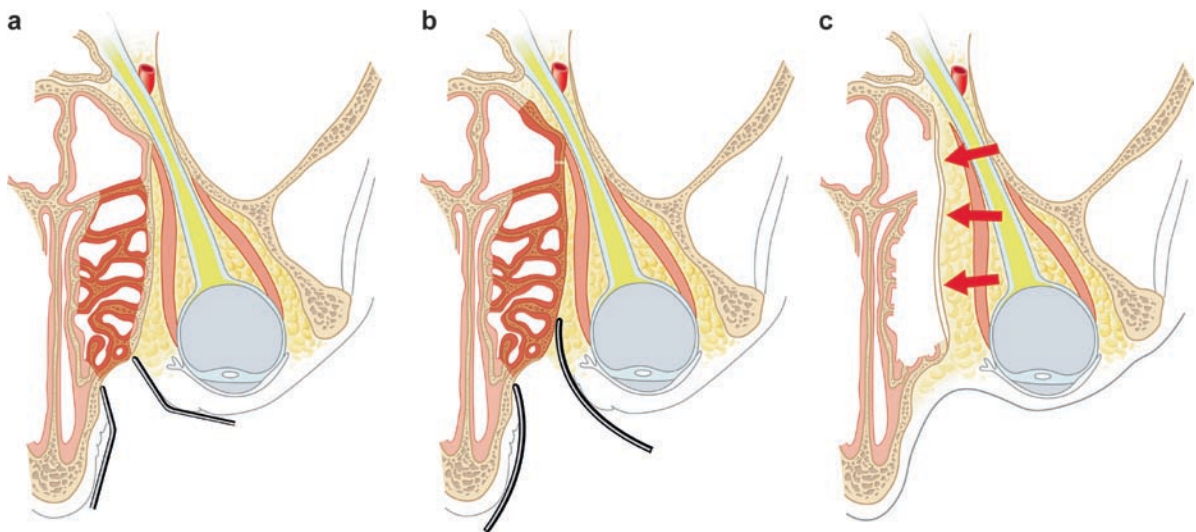


Fig. 6.10 (a) Transfacial approach for transtethmoidal-transsphenoidal decompression of the optic nerve and the orbital cavity (mod. a. Weerda 1995). (b) Decompression of the optic nerve by ethmoidectomy and removal of the lateral wall of the sphenoid

wing (red: removed bony structures). (c) Endonasal/transethmoidal decompression of the orbit and the optic nerve after ethmoidectomy, resection of the orbital lamina of the ethmoid (lamina papyracea) and of the anterior and lateral walls of the sphenoid

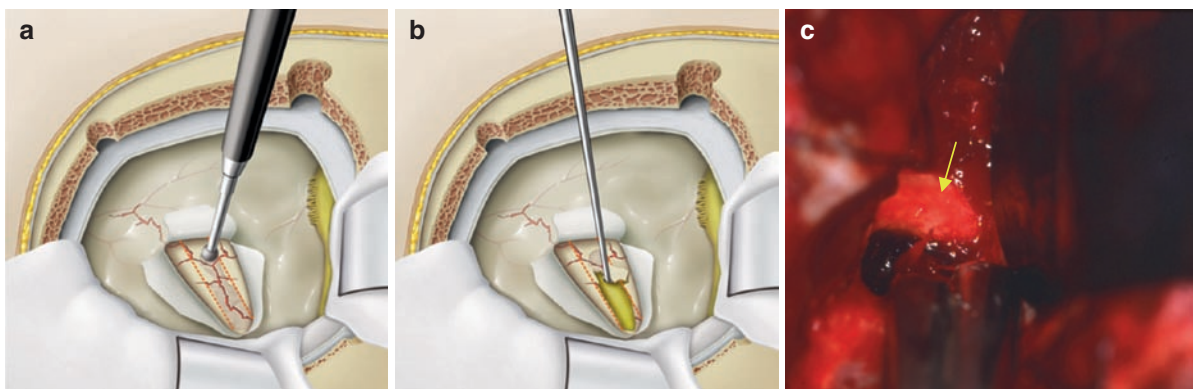


Fig. 6.11 Transcranial decompression of the optic nerve (mod. a. Kastenbauer and Tardy 1995). Incision of the dura (a). Microscopic debridement with removal of the fragments of the

roof of the optic canal. (b) Incision of the optic nerve sheath. (c) Intraoperative view during transcranial exploration of the optic nerve (arrow) after CCMF fracture

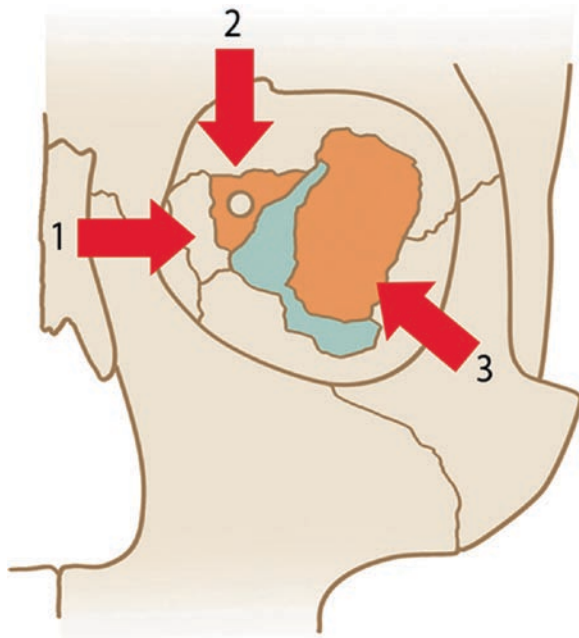


Fig. 6.12 Biomechanical vectors in midface trauma. The vectors are diverted by the orbital walls and converge to the apex of the orbit (mod. a. Hardt and Steinhäuser 1979). 1 Complex central midface fracture/central naso-orbito-ethmoidal fracture, 2 cranio-orbital fracture/frontobasal fracture, 3 complex lateral midface fracture

6.1.2.2 Injuries at the Cranio-Orbital Junction

Midface fractures on a high level in combination with orbital wall fractures can cause serious mechanical and neurogenic ophthalmologic complications (Herrmann

1976; Holt and Holt 1983; Brent and May 1990; Al-Qurainy et al. 1991a, c; Hardt and Sgier 1991; Fonseca and Walker 1991; Soparkar 2005).

Fractures in the roof of the orbit, especially in the region of the cranio-orbital junction are caused by dislocated lateral midface fractures running through the greater wing of the sphenoid, by cranial fractures with depression of fragments and also by complex central midface fractures through the lesser wing of the sphenoid (Hardt and Steinhäuser 1979; Leider and Mathog 1995) (Fig. 6.12).

Fractures of the greater and lesser wing of the sphenoid can traumatize the cranial nerves III to VI, as they pass through the skull base into the orbit. The optic nerve may also be damaged to a varying degree (Manfredi et al. 1981; Ghobrial et al. 1986; Hardt and Sgier 1991; Leider and Mathog 1995).

Neurological damage is caused by direct compression, by bony fragments or by an indirect compression of the nerves caused by hemorrhage (Rowe and Williams 1985).

Reduced motility of the globe (nerves III, IV, VI) results from fractures in the area of the superior orbital fissure or the roof of the orbit and can lead to rare, but typical ophthalmologic syndromes (Kretschmer 1978; Fonseca and Walker 1991; Dutton and Al-Qurainy 1991) (Fig. 6.13).

Based on the neurological deficits, different orbital syndromes can be distinguished (Hardt and Steinhäuser 1979; Hardt and Sgier 1991; Dutton and Al-Qurainy 1991):

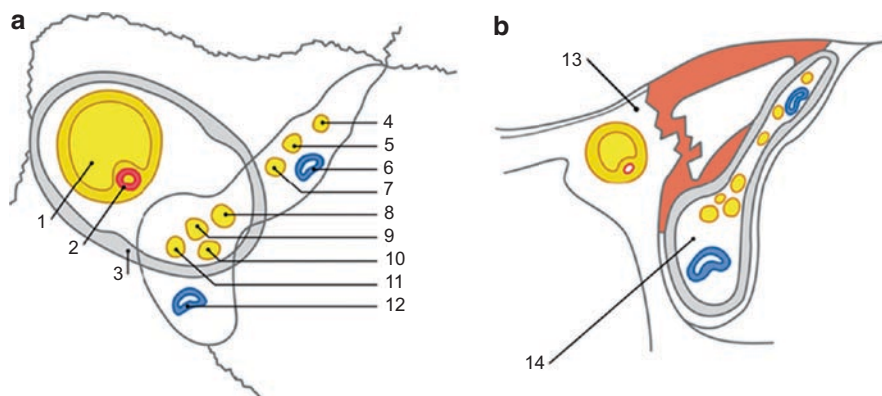


Fig. 6.13 (a) Coronal section through the superior orbital fissure (with entering cranial nerves and vessels) and the lesser wing of sphenoid (with optic canal) (mod. a. Ghobrial et al. 1986, Hardt and Sgier 1991). (b) Compression of nerves and vascular structures of the superior orbital fissure by displaced bone fragments of the lesser wing of the sphenoid. 1 Optic

nerve, 2 ophthalmic artery, 3 annulus tendinosus (Zinns tendon), 4 lacrimal nerve, 5 frontal nerve 6 superior ophthalmic vein, 7 trochlear nerve, 8 oculomotor nerve, 9 nasociliary nerve, 10 abducent nerve, 11 oculomotor nerve, 12 inferior ophthalmic vein, 13 lesser wing of sphenoid, 14 superior orbital fissure

- Partial or complete superior orbital fissure syndrome
- Retrobulbar hemorrhagic compression syndrome
- Orbital apex syndrome
- Clivus syndrome

Frequency

The frequency of orbital syndromes in complex mid-face fractures is approximately 8% (Hardt and Sgier 1991).

Most of these syndromes are superior orbital fissure syndromes (SOFS) and hemorrhagic compression syndromes (HCS), occurring in 2.2%. The nervus opticus syndrome (NOS) follows at 1.9%, the orbital apex syndrome (OAS) at 1.6% and the sinus cavernosus syndrome (SCS) at 0.3% (Hardt and Sgier 1991).

Percentage of orbital syndromes in complex midface fractures (Hardt and Sgier 1991)

Superior orbital fissure syndrome (SOFS)	2.2%
Hemorrhagic-compression syndrome (HCS)	2.2%
Nervus opticus syndrome (NOS)	1.9%
Orbital apex syndrome (OAS)	1.6%
Sinus cavernosus syndrome (SCS)	0.3%

Distinction between the syndromes according to fracture types show that in most cases the OFS and the HCS are caused by lateral and also centrolateral mid-face fractures. The OAS and the NOS result mostly from isolated or central midface fractures with cranio-orbito-ethmoidal fractures and penetration injuries (Hardt and Sgier 1991) (Fig. 6.14).

Superior Orbital Fissure Syndrome (SOFS)

Dislocated bony fragments or comminuted fractures in the region of the superior orbital fissure or of the lesser wing of the sphenoid cause direct nerve lesions. If all cerebral nerves entering at this point are involved a complete superior orbital fissure syndrome results.

The Complete SOFS

The complete SOFS results from a paresis of the cerebral nerves III, IV, and VI. Clinically there is an ophthalmoplegia with ptosis and an exophthalmus due to disruption of the venous drainage. In addition, a mydriasis and an accommodation paralysis (cycloplegia) occur due to loss of the parasympatic innervation.

Fig. 6.14 Relation and frequency of orbital syndromes in different types of midface fractures (Hardt and Sgier 1991). 1 Central midface fracture, 2 centrolateral midface fracture, 3 lateral midface fracture, 4 cranio-orbital and midface fracture, 5 cranio-orbito-ethmoidal fracture, 6 penetrating injuries

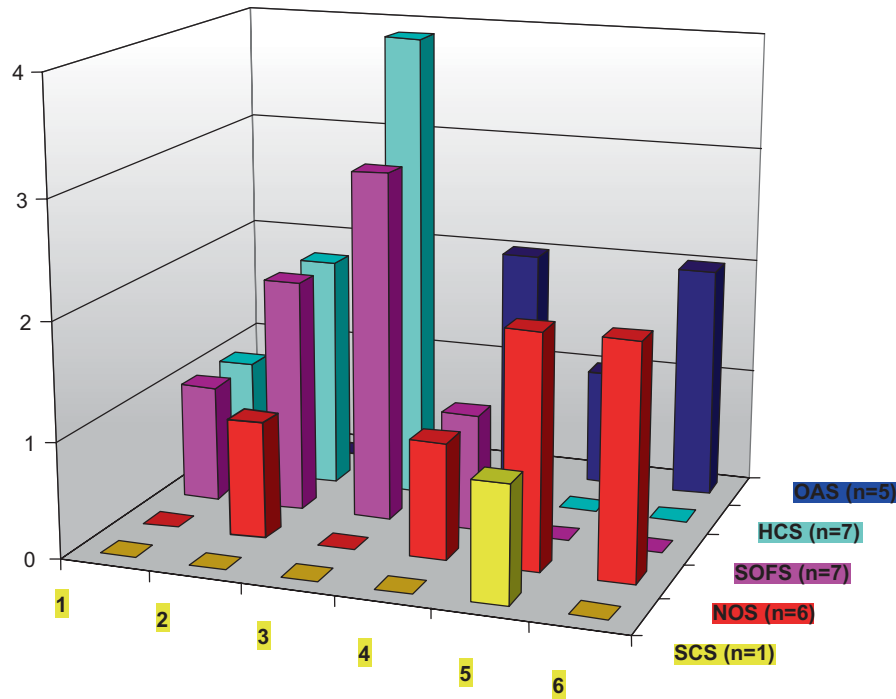




Fig. 6.15 Complete right upper orbital fissure syndrome with palsy of cranial nerves III, IV, and VI following lateral midface fracture and fracture of the greater wing of the sphenoid (6-weeks post surgery)

If the abducent nerve is still intact, abduction of the eyeball may still be possible (Hedstrom et al. 1974).

Anesthesia in the areas of sensory innervation is inevitable if the sensory branches of the ophthalmic nerve are involved. A severe retroorbital pain sometimes occurs in combination with a supraorbital neurogenic pain as a result of the complex damage that has occurred (Hardt and Sgier 1991).

As a consequence of fragment dislocation, decompression of the orbital fissure via the fronto-temporal access is necessary (Mathog et al. 1995) (Fig. 6.15).

Symptoms

- Complete ophthalmoplegia (nerves III, IV, VI)
- Ptosis
- Mydriasis - accommodation paralysis (cycloplegia)
- Loss of sensibility (ophthalmic, nasociliary nerve)
- Retroorbital pain
- Possible abduction of the eyeball

Incomplete SOFS

Due to the fact that three oculomotor nerves enter through the superior orbital fissure, injuries to the individual branches may lead to a selective paresis, so developing the image of a partial SOFS. Frequent injuries occur in the caudal region of the superior orbital fissure. The clinical loss is usually limited to restriction of the vertical motility of the eyeball due to partial damage of the nerves III and IV, a ptosis, an abnormal pupil reaction and anesthesia in the innervation area of the nasociliar nerve (Hardt and Sgier 1991). Isolated damage to the parasympathic element of the third cerebral nerve leads to a temporary mydriasis, this occurs relatively often in lateral midface fractures (Fig. 6.16).

Symptoms

- Partial ophthalmoplegia (nerves III and/or IV)
- Ptosis
- Mydriasis
- Accommodation paralysis (cycloplegia)

Hemorrhagic Compression Syndrome (HCS)

Massive retrobulbar hemorrhage in the posterior region of the muscle cone, triggered by vessel disruption, leads to progressive exophthalmus with concurrent pupil dilatation, reduced vision and increased intraocular pressure (Ord 1981; Ord and El Altar 1982).

This is a consequence of complex lateral and centro-lateral midface fractures and rarely of dislocated fractures in the region of the orbital apex (Hardt and Sgier 1991). Hemorrhage, which causes an increase in intra-orbital pressure results in indirect nerve injuries, especially to the optic nerve. These injuries can only be prevented by speedy decompression of the retroorbital hematoma (Rowe 1977; Alper and Aitken 1988; Hardt and Sgier 1991) (Figs. 6.17–6.19).

The following are typical signs of an intraorbital hemorrhage with or without orbital fracture (Doden and Schnaudigel 1978):

- Livid (cyanotic) swollen eyelids with narrow spontaneous palpebral lid opening, which may be opened actively, though passive opening is only slight
- Protrusion of the globe (up to 10 mm) with increasing active and passive immobility
- Increased intra-ocular pressure up to 80 mmHg (normal pressure 20 mmHg)
- Ischemia of the optic disk and retina with clearly reduced vision or amaurosis
- General symptoms similar to the Aschner-symptom-complex (bradycardia, nausea, sweating caused by vagal impulses)

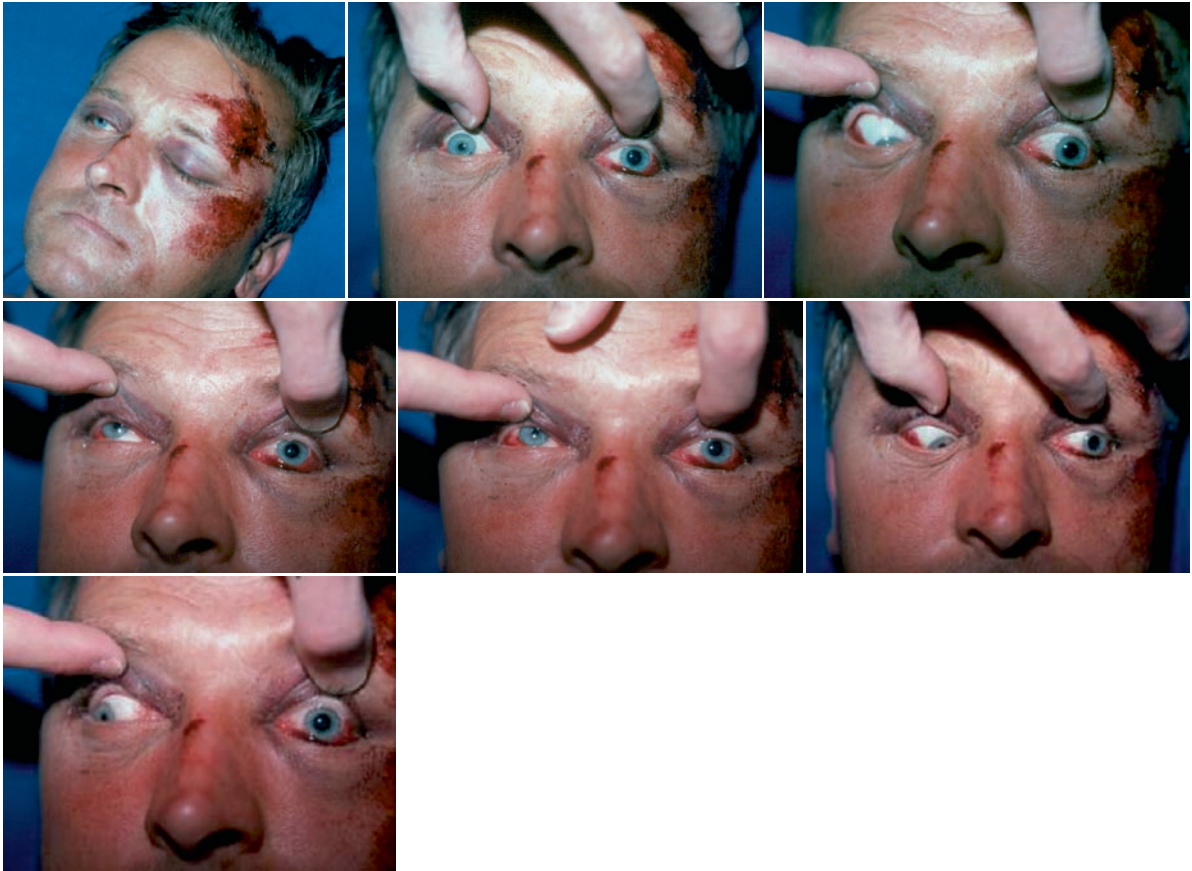


Fig. 6.16 Incomplete left superior orbital fissure syndrome with palsy of the oculomotor nerve. Mydriasis, partial ophthalmoplegia, ptosis, and exophthalmia following fractures of the zygoma and sphenoid wing

Symptoms

- Complete/incomplete ophthalmoplegia
- Progressive, massive exophthalmus
- Mydriasis
- Vomiting (oculo-gastral reflex)
- Inwards shifting of the orbital wall (blow-in fracture)
- Subperiosteal hematoma (persistent), leading to a reduction of the intraorbital volume and exophthalmus
- Orbital compartment syndrome (any sudden increase in orbital pressure, mostly caused by acute arterial bleeding (ethmoidal artery) or an emphysema)

It is essential to decompress all retrobulbar hematomas with reduced vision, ischemia of the optic papilla and retina, with high intraocular pressure and progressive or manifest exophthalmus in order to prevent permanent damage to the optic nerve (Krausen et al. 1981; Gellrich 1999). The decompression is performed via the transfacial-latero-orbital or transfacial-tranethmoidal approach (Hardt and Sgier 1991; Mathog 1992; Rochels and Rudert 1995) (Fig. 6.20).

Compression in the region of the orbital apex can also be caused by:

Orbital Apex Syndrome (OAS)

If retroorbital fractures not only affect the superior orbital fissure, but lead to an immediate lesion of the optic nerve an OAS develops (Brent and May 1990; Radtke and Zahn 1991).

In this case, a high-grade loss of visual acuity or an amaurotic complete iridoplegia occurs. In addition, there is paresis of the three cranial nerves III, IV, and VI (Lisch 1976; Hardt and Sgier 1991; Stewart and Soparkar 2005; Soparkar 2005) (Fig. 6.21).

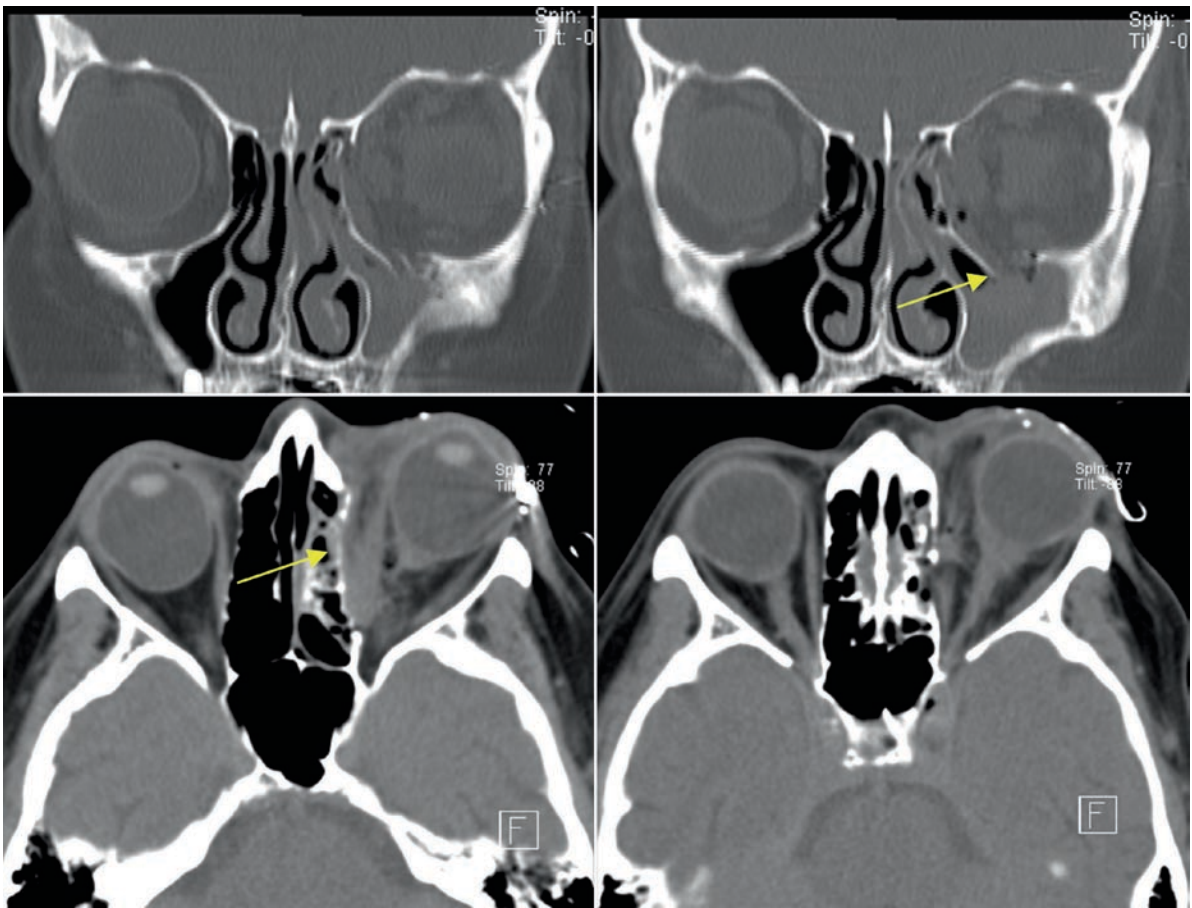


Fig. 6.17 Intraconal hemorrhage after contusion injury to the orbit with fracture of the medial and inferior orbital walls (*arrow*). Bleeding at the posterior surface of the orbit and along the optic nerve. Hematoma along the medial orbital wall



Fig. 6.18 Hemorrhagic retrobulbar compression syndrome with exophthalmos and ophthalmoplegia after contusion injury to the orbit with depression fracture of the orbital floor (*arrow*)

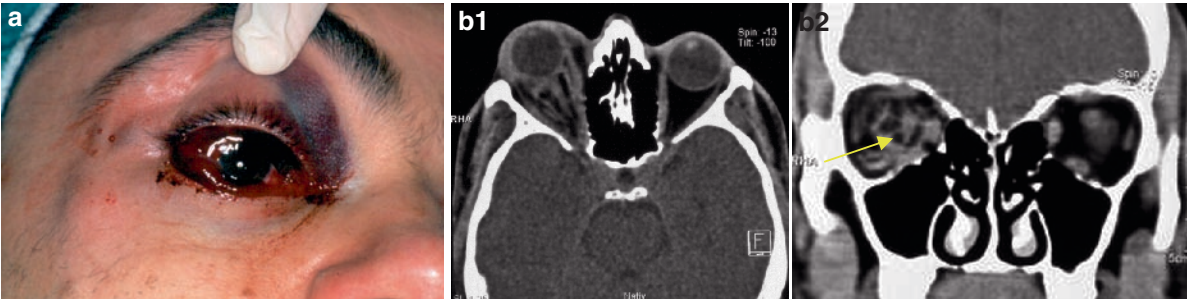


Fig. 6.19 Contusion injury to the globe without orbital fracture, but with significant diffuse retrobulbar hemorrhage. (a) Retroorbital hemorrhage with consecutive exophthalmus and ophthalmoplegia. (b) CT: diffuse retrobulbar hemorrhage with exophthalmus (arrow)

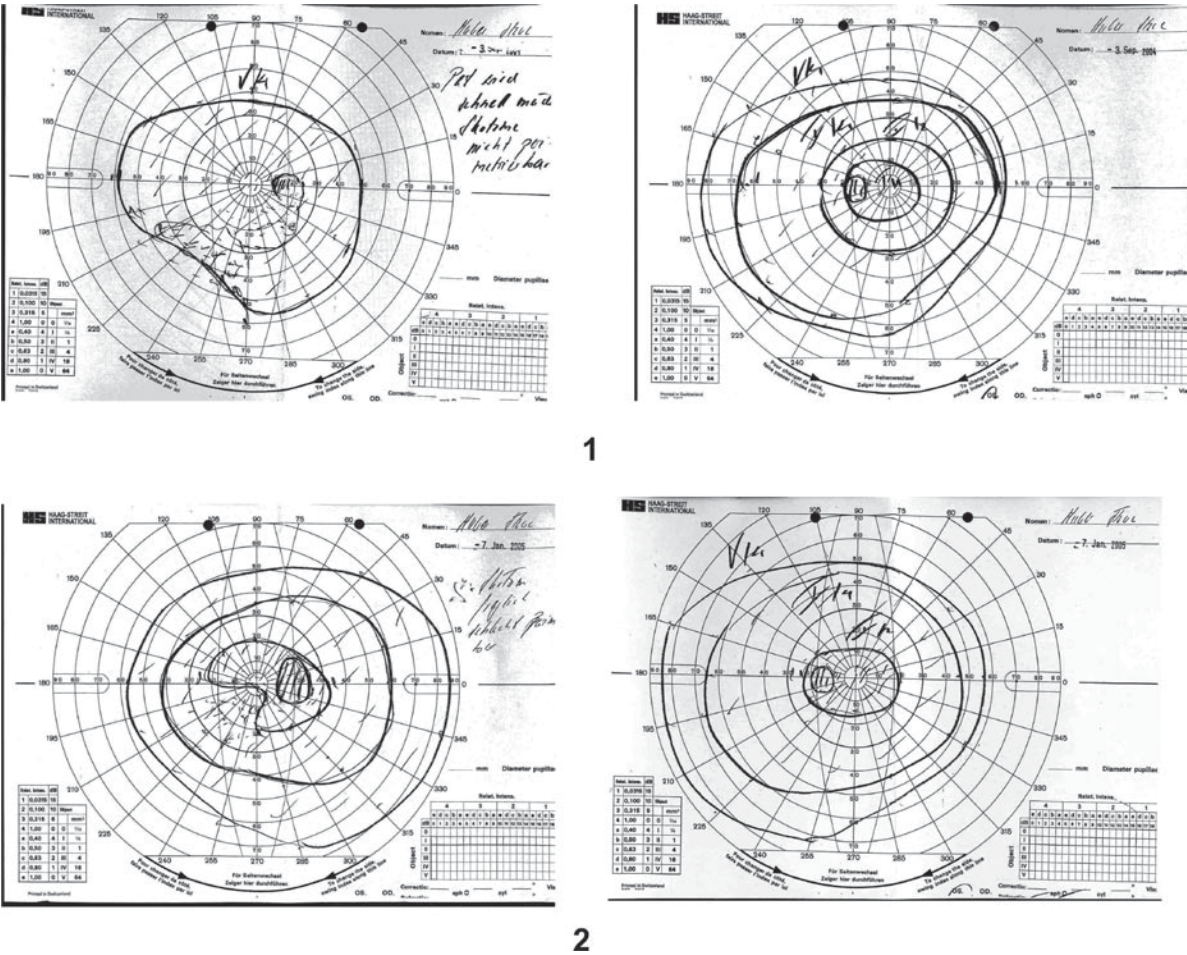


Fig. 6.20 Course of retroorbital compression syndrome after fracture of the zygoma and the greater sphenoid wing. 1 Visual field immediately after surgical decompression of the orbit; 2 visual field at 3-months follow-up



Fig. 6.21 Orbital apex syndrome with internal ophthalmoplegia and amaurosis and complete iridoplegia following zygomatico-orbital fracture on the right

Symptoms

- Loss of function of cranial nerves III, IV, VI
- Damage of the optic nerve (N. II) with loss of visual acuity

Clivus Syndrome

Intracranial bleeding can lead to a compression and interruption of the oculomotor nerve at the clivus prior to its point of entrance into the superior orbital fissure. The clinical consequence is a mydriasis on the affected side.

6.1.2.3 Vascular Injuries in Skull Base Trauma

Particularly endangered in frontal skull base injuries are the cavernous sinus and the sagittal sinus. There is potential injury to the internal carotid artery by fractures in the sphenoid region, especially in the pyramidal apex. Carotid-cavernous sinus fistulas are more common than isolated injuries to the internal carotid artery.

Cavernous Sinus Syndrome

In severe craniofacial trauma with fractures of the sphenoid and/or petrous bone, there may also be an injury to the internal carotid artery on its course through the cavernous sinus (Takenoshita et al. 1990). As a consequence of arterial damage, arterial blood flow into the venous circulatory system occurs (cavernous arterio-venous fistula), producing a back-flow in the orbital veins (superior ophthalmic vein) (Hasso et al. 1979; Harris et al. 1984).

Clinically, this results in a massive unilateral (occasionally bilateral and seldomly contralateral) pulsating, progressive exophthalmus (exophthalmus pulsans)

and leads to an extreme conjunctival reddening through intense dilatation and congestion of the conjunctival and episcleral veins. There is additional chemosis and lid swelling.

This results in a pulse synchronic sound above the eye and beside the temple and cerebral nerve damage (abducent nerve, oculomotor nerve, trochlear nerve, first, and second trigeminal branches) due to their course within the cavernous sinus wall as well as reduction in visual acuity and visual field. Further risks arise from raised intraocular pressure.

The reduction of vision may end in a complete amaurosis. Vascular fistulas can be detected by MRI examination as well as by angiography (Schaller 2002) (Fig. 6.22).

Symptoms

- Internal ophthalmoplegia
- Exophthalmus (pulsating)
- Ptosis
- Conjunctival injections
- Pulse synchronic sound

Thrombosis of the Superior Ophthalmic Vein

Fractures in the region of the orbital roof can lead to a thrombosis of the superior ophthalmic vein, resulting in a mild exophthalmus and damage to the external branch of the oculomotor nerve. Ptosis and reduced motility are clinical signs.

Craniofacial injuries can also lead to disruptions of the ophthalmic and the anterior cerebral arteries as well as the anterior and posterior ethmoidal arteries. In frontal skull base injuries the cavernous sinus and the sagittal sinus are particularly susceptible to lacerations. Thrombi, hemorrhage, and air embolism are possible consequences (Fig. 6.23).

Fig. 6.22 Carotis-sinus-cavernosus-fistula in a lateral zygomatico-orbital and sphenoidal fracture on the right. (a) Right internal ophthalmoplegia, exophthalmus, ptosis, and conjunctival injection. (b) Axial CT image with i.v. contrast injection: large superior orbital vein and cavernous sinus on the right (arrow)

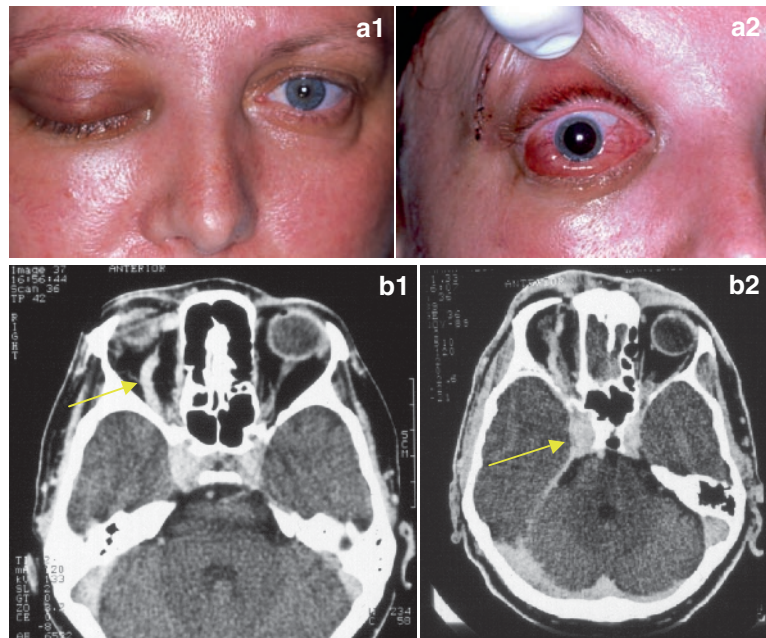
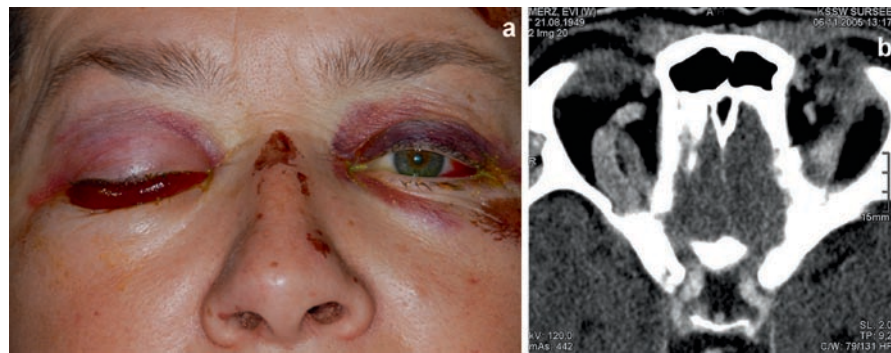


Fig. 6.23 Thrombosis of superior orbital vein after right orbital fracture. (a) Exophthalmus, conjunctival edema, ptosis, and significant restriction of eye motility. (b) MRI (fat-suppressed T1-weighted images after contrast injection): dilatation and central thrombus in the right superior orbital vein



6.1.3 Questionable Signs of Skull Base Fractures

6.1.3.1 Hematoma Around the Eyes/Lid Hematoma

There is seldom an absence of a monocular or binocular hematoma following skull base injuries, though this is not necessarily conclusive as hematomas may just as well occur following direct trauma or midface injury.

Lid hematomas may occur as a result of orbital roof or craniofacial injuries. In this case the uni- or bilaterally swollen, initially blue-colored upper lids are quite impressive. Lid elevation is impaired or impossible. The orbital septum separates the upper lid into compartments and provides a possibility to differentiate between midface fractures and frontal skull base fractures. The examination is carried out by ectropionizing the eyelid.

- *Facial skeletal injuries* only exhibit hematomas ventral to the orbital septum, which are visible shortly after the accident.
- *Skull base injuries* exhibit lid hematomas dorsal and ventral to the orbital septum. There may be a delay following injury before the hematoma becomes evident due to the longer distance from the skull base to the lid.

6.1.3.2 Hemorrhage in the Skull Base Region

Basal Mucosal Hemorrhage

Mucosal injuries in the paranasal sinuses adjacent to the skull base caused by fractures may result in temporary, intensive hemorrhage from the nose. As a rule hemorrhaging from extrabasal mucosal tears is self-obliterating.

Hemorrhage in Frontal Skull Base Fractures

The ethmoid artery and branches of the maxillary artery are involved in extensive ethmoid fractures. If there is no spontaneous obliteration, there may be heavy nasal and pharyngeal bleeding, which will require intervention.

Massive bleeding which spreads through the fractured medial orbital wall into the posterior orbit may cause exophthalmus and gradual compression of the optic nerve and the ophthalmic artery.

The result is a *hemorrhagic compression syndrome* with progressive loss of vision. Similar complications can be expected if hemorrhaging from the ethmoid arteries is inappropriately controlled, obliterating the apparent nasal hemorrhaging, yet not sufficiently compressing the arterial stump.

Fractures of the middle cranial fossa mostly result in hemorrhaging from the ear or into the tympanic cavity (hematotympanon) with the result of possible deafness.

6.3.1.1 Emphysema

Subcutaneous emphysema indicates fractures of the walls of the paranasal sinuses. This is a regular finding in ethmoid fractures and can be an indication of skull base involvement.

Orbital Emphysema

Orbital emphysema (*intraorbital emphysema*) suggests a frontal skull base or ethmoid fracture with mucosal tearing, or an orbital floor fracture. If air escapes from the nose and paranasal sinuses, via the orbit into the lid system, a *lid emphysema* results, which can be recognized by so-called emphysema crackling and is partly associated with simultaneous *conjunctival emphysema*.

6.2 Midface Injuries (Clinical Signs)

Whilst clinically examining the midface one should seek mobility of the maxillary block with possible dislocation (dorso-caudal displacement/disturbed occlusion), an open bite and bony steps along the periorbital rim,

the zygomatic bone, the nasal skeleton and intraorally in the upper vestibule along the zygomatico-maxillary buttress (Lew and Sinn 1991; Keith 1992; Schwenzer and Ehrenfeld 2002).

Direct signs of midface fractures are

- Abnormal motility
- Dislocation
- Abnormal occlusion
- Evidence of crepitation and abnormal resonance

6.2.1 Central Midface Fractures without Abnormal Occlusion (NOE Fractures)

In central midface fractures, depression of the nasomaxillary struts, fractures of the medial orbital wall and naso-ethmoidal structures may lead to an increase in distance between the medial angles of the eyes (telecanthus). Avulsion of the canthal ligaments, increasing interpupillary distance (hypertelorismus) and injuries to the nasolacrimal duct are possible.

Simultaneous fractures of the frontal skull base with dural lacerations may result in rhinoliquorrhea and anosmia caused by avulsion of the olfactory filaments (Holt 1986).

6.2.2 Central Midface Fractures with Abnormal Occlusion (Le Fort I and II)

The most important sign is the abnormal mobility of the fractured midfacial block. Maxillary displacement regularly results in malocclusion. Dorsal and caudal displacement of the midface results in an anterior open bite as well as flattening and lengthening of the midface.

Uneven surfaces can be palpated on the anterior bony aperture of the nose, the facial wall of the maxillary sinus and particularly on the zygomatico-alveolar buttress and in the region of the infraorbital margin. In the rare case of ruptured retromaxillary vessels (maxillary artery and vein) life-threatening hemorrhage may occur (Spiessl et al. 1976).

Sensory disorders (hyp-/anaesthesia) occur regularly in the region supplied by the infraorbital nerve as

a consequence of infraorbital foramen or infraorbital canal involvement.

6.2.3 Centrolateral Midface Fractures with Abnormal Occlusion (Le Fort III)

The clinical symptoms of Le Fort II and III fractures are similar. The midface is abnormally mobile, flattened, and displaced in a dorsal and caudal direction. Malocclusion is a regular feature. Uneven surfaces in the region of the lateral orbit and root of the nose are often present, but not always evident due to swelling. Skull base involvement with liquorrhea as a result of dural injury is not uncommon. Additional dural injuries in polytraumatized patients lying in a supine position are initially not clinically obvious, as the liquor runs directly into the nasopharynx and, at best, the patient's frequent swallowing becomes conspicuous.

6.2.4 Lateral Midface Fractures

A monocular hematoma and depression in the zygomatic region are often clinically impressive. Depending on fracture type, there are often palpable steps alongside the lateral orbit, at the infraorbital margin and at the zygomatico-alveolar buttress.

In strongly dislocated fractures and comminuted fractures, the facial prominence is flattened. Extreme zygomatic displacement results in reduced mouth opening. In case of extensive dislocation of the orbital floor, there may be an additional difference of the pupillary axis and disturbed eye motility resulting from ocular muscle entrapment as well as an enophthalmus (Spiessl and Schroll 1972).

As the infraorbital foramen and the infraorbital canal are almost constantly involved in zygomatic fractures, there are frequent sensory defects of the infraorbital nerve (Spiessl and Schroll 1972; Schwenzer and Ehrenfeld 2002) (Fig. 6.24).

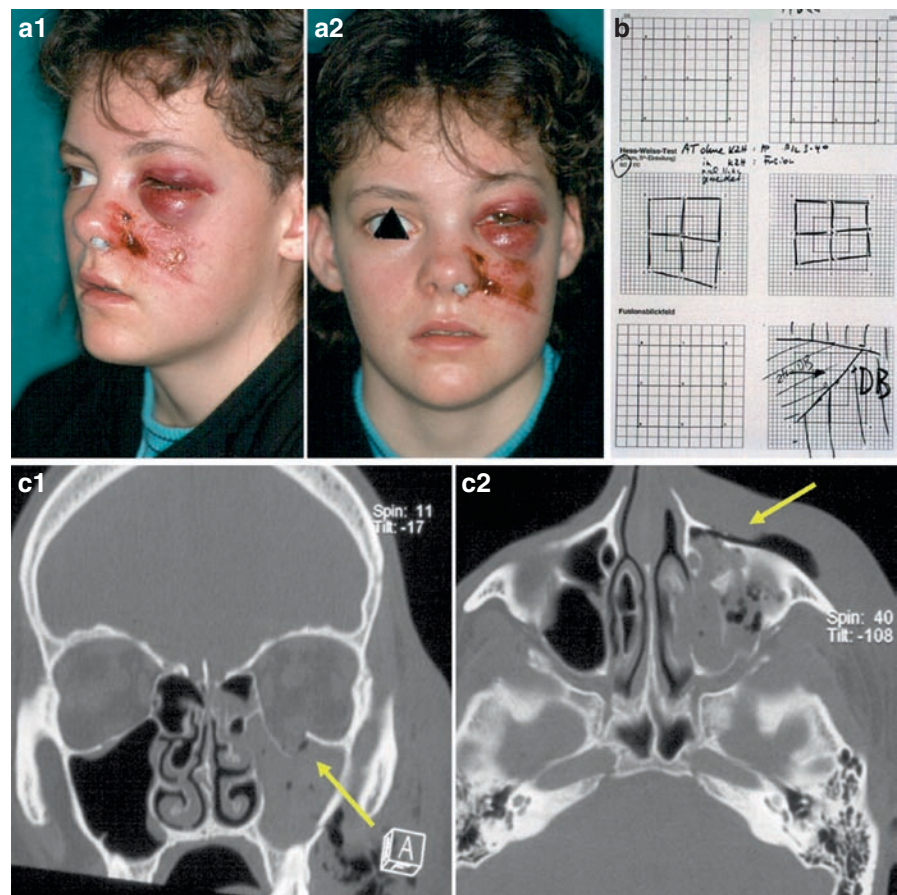


Fig. 6.24 Disturbance of motor coordination by impaired eye motility after displaced fracture of the lateral midface with depression of the zygoma and displacement of the ocular muscles. (a) Photographs. (b) Hess-Weiss test. (c) Coronal and transverse CT images with depression of the orbital floor and inward displacement of the zygoma (arrow)

6.3 Orbital Injuries

6.3.1 Orbital Soft-Tissue Injuries

In 40–70% of craniofacial traumas, there are additional orbital injuries [Hardt and Sgier 1991 (47%); Kramp et al. 1997 (72%)].

Orbital soft-tissue injuries (Jabaley et al. 1975; Flick 1976; Ioannides et al. 1988; Dutton and Al-Qurainy 1991; Brandes et al. 1997; Kramp et al. 1997; Brown et al. 1999; Poon et al. 1999; Rohrbach et al. 2000) in midface traumas include:

6.3.1.1 Minor Eye Injury

Subconjunctival hemorrhage, hyposphagma, ecchymosis, corneal-injuries.

6.3.1.2 Nonperforating Injury of the Globe

Contusion/distorsion of the globe, iridodialysis, lens luxation, retinal detachment and edema of the optical nerve sheath (Fig. 6.25).

6.3.1.3 Perforating Injury of the Globe (2%)

Perforation, rupture or loss of the globe (Fig. 6.26).

6.3.1.4 Traumatic Neuropathy [Posttraumatic Optic Neuropathy (PTON)]

In direct frontal orbital contusions, the compression wave is concentrated in the orbital apex. The nervous structures cannot evade the blow and are damaged by the pressure and consequent edema, resulting in optic neuropathy (Rochels and Behrendt 1996; Ernst et al. 2004).

The frequency of such traumatic neuropathies (PTON) in facial skeletal injuries is quoted to be 3.6% (Joseph et al. 1990; Dutton and Al-Qurainy 1991) (Fig. 6.27).

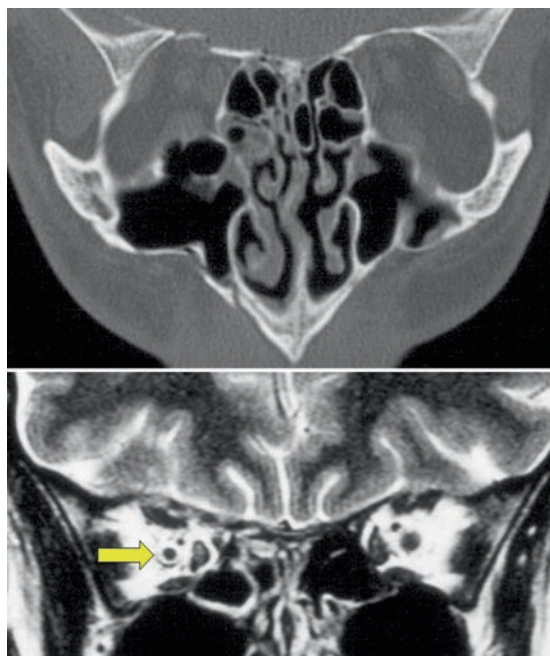


Fig. 6.25 Edema of optical nerve sheath (arrow) after orbital roof fracture and contusion of the globe

6.3.2 Orbital Wall Fractures

In order of frequency, orbital wall fractures occur particularly in combination with the following fracture complexes (Paskert et al. 1988; Fonseca and Walker 1991; Manson 1998; Manolidis et al. 2002):

- Zygomatico-orbital fractures
- Naso-orbito-ethmoid fractures
- Complex upper midface fractures

6.3.2.1 Fracture Frequency

Orbital wall fractures are found in 9.2% of all cranio-cerebral/frontobasal fractures and in 30–55% of all midface fractures (Neubauer 1987; Holt and Holt 1983; Fonseca and Walker 1991).

Fig. 6.26 Perforation injury of the right orbital globe (ophthalmorrhexis) after tangential trauma to the lateral midface

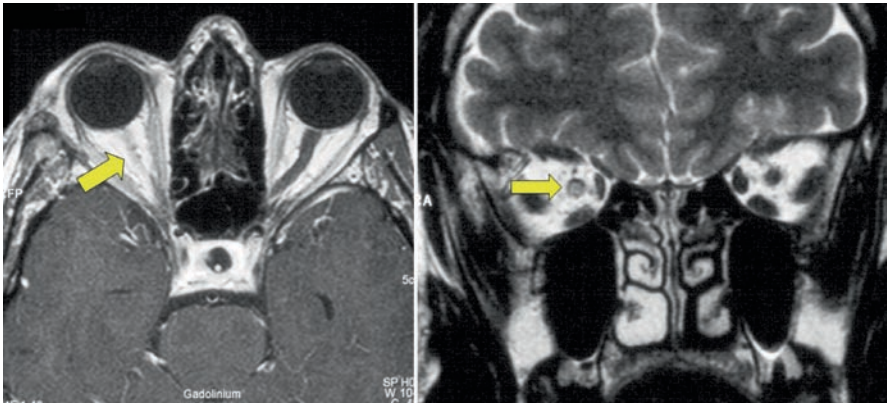
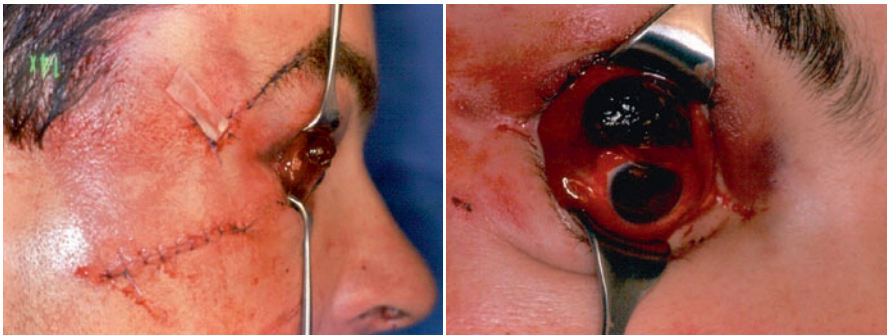


Fig. 6.27 MRI of posttraumatic optic neuropathy (PTON). (a) Coronal T2-weighted image with swelling and elevated T2-signal in the right optic nerve. The low signal spot in the center of the nerve represents the optic artery (*arrow*). (b) Transverse T1-weighted

image after i.v. contrast medium injection. Normal low signal of the left optic nerve. High signal of the right optic nerve representing intense contrast enhancement (*arrow*). The enhancement indicates posttraumatic inflammation of the nerve

Frequency of isolated and combined orbital wall fractures in midfacial trauma (Kramp et al. 1997)

Isolated orbital wall-fractures	4–16% of all facial fractures
Combined midface/orbital wall fractures	30–55% of all facial fractures

According to Kramp et al. (1997) the orbital floor is involved in 69% of all orbital fractures, the medial orbital wall in 19% and combined orbital floor and medial wall fractures in 10%.

Less common are orbital roof fractures at 1.2% and combined medial orbital wall/orbital floor/orbital roof fractures with a frequency of 0.6%.

Orbital wall involvement in midfacial fractures (Kramp et al. 1997)

Orbital floor	69%
Medial orbital wall	19%
Medial orbital wall + orbital floor	10%
Orbital roof fractures	1.2%
Medial orbital wall + orbital roof + orbital floor	0.8%

6.3.3 Fracture Localization

6.3.3.1 Orbital Floor Fractures

In midface trauma, orbital floor fractures may be isolated or combined with additional midface fractures. On average, 72% of the orbital floor fractures show varying degrees of fragmentation, whereas 28% show only fissures without dislocation. The medio-nasal and central floor segments are usually affected (Hawes and Dortzbach 1983; Kramp et al. 1997).

Distribution of orbital floor fractures with simultaneous midfacial fractures (Kramp et al. 1997)

Entire orbital floor	62%
Segments of the orbital floor	38%
Medial orbital floor	51%
Central orbital floor	42%
Lateral orbital floor	7%

Ophthalmological symptoms are found in 40% of orbital floor fractures (Dutton and Al-Qurainy 1991). Depending on fracture mode, there may be *muscle or ligament entrapment* in the fracture gap as well as

injury to the orbital muscles with consequent disturbances of eye motility or altered position of the globe (Flanagan et al. 1980; Koornneff 1982, 1987; Joss 1995) (Figs. 6.28–6.34).

Fig. 6.28 (a) The inner ligament and suspensory system in the periorbital fat of the eye. (b) Comminuted blow-out fracture of the orbital floor, resulting in herniation of periorbital fat into the maxillary sinus with consecutive enophthalmus. Dysbalance of the globe, incarceration of the radial periorbital ligaments and indirect restriction of the movement of the inferior rectus muscle (mod. a. Rowe and Williams 1985; Manson et al. 1986)

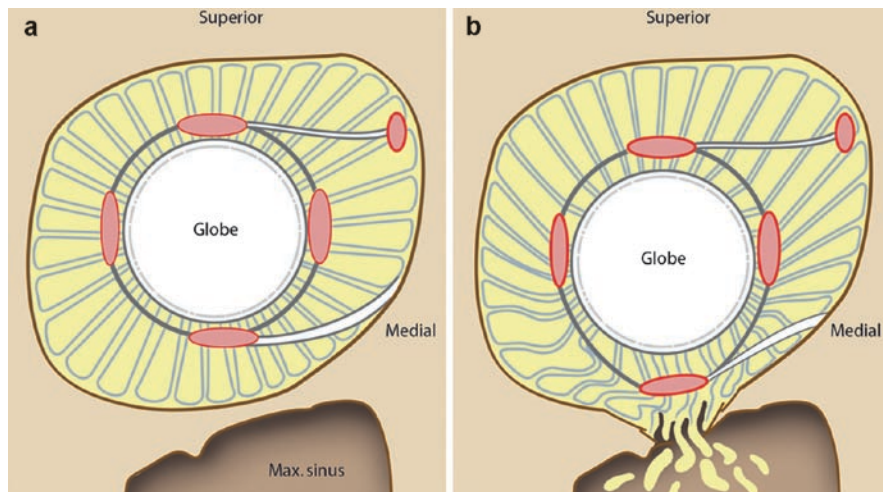


Fig. 6.29 Blow-out fracture of the orbital floor. Door-wing-like displacement of the medial half of the orbital floor into the maxillary sinus. Herniation of orbital fat into the sinus

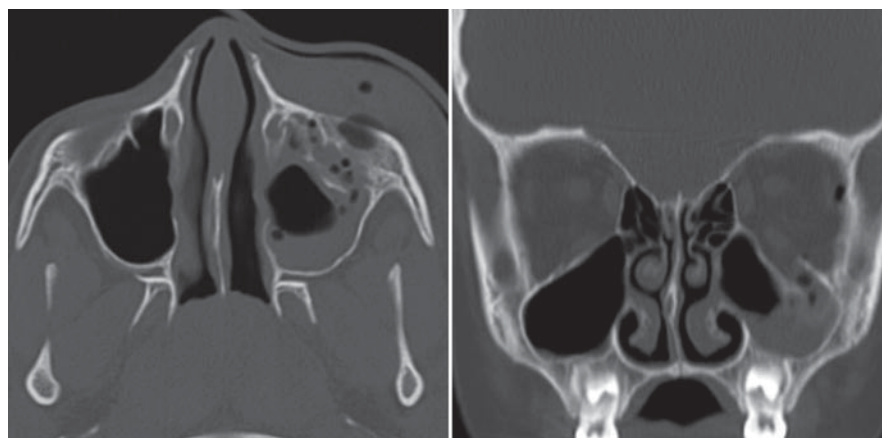
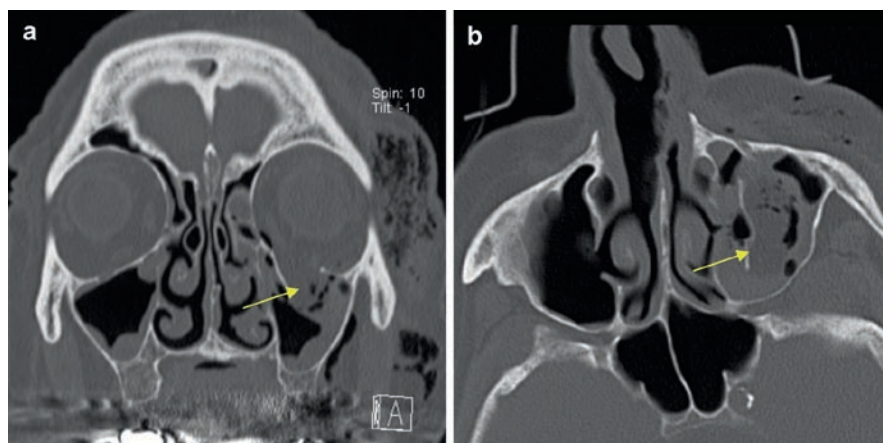


Fig. 6.30 Blow-out fracture of the orbital floor. (a) Coronal CT image shows door-wing-like displacement of the medial half of the orbital floor into the maxillary sinus. Herniation of fat into the sinus (arrow). (b) The transverse image demonstrates the bone fragment within the sinus (arrow)



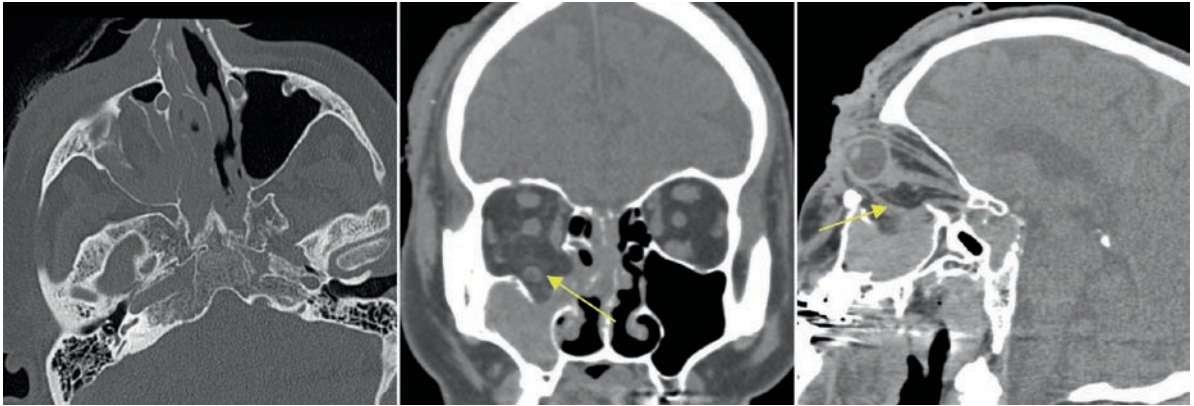


Fig. 6.31 Displaced right orbital floor fracture with herniation of bone and fat into the maxillary sinus. Bow-like downward displacement of the inferior rectus muscle (*arrow*)

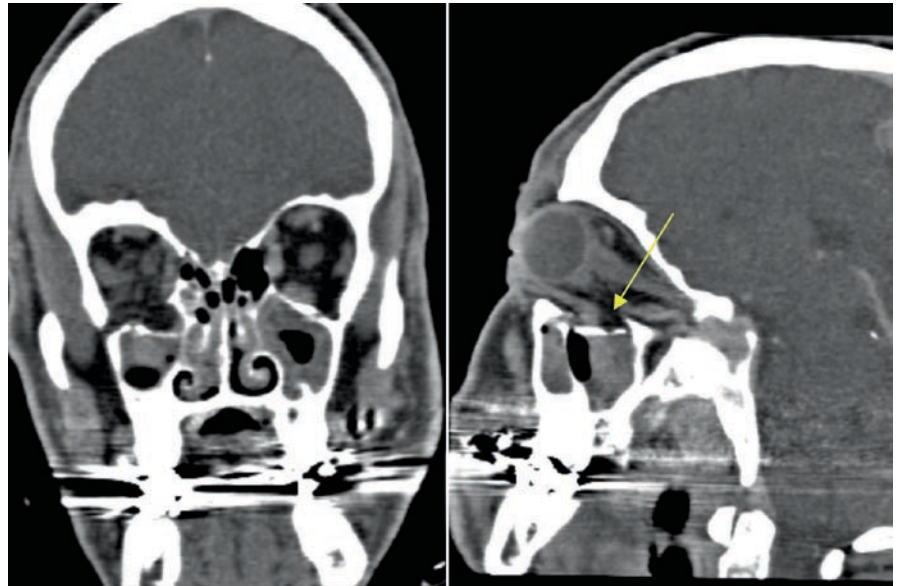


Fig. 6.32 Blow-out fracture of the orbital floor with disruption of the inferior rectus muscle (*arrow*)

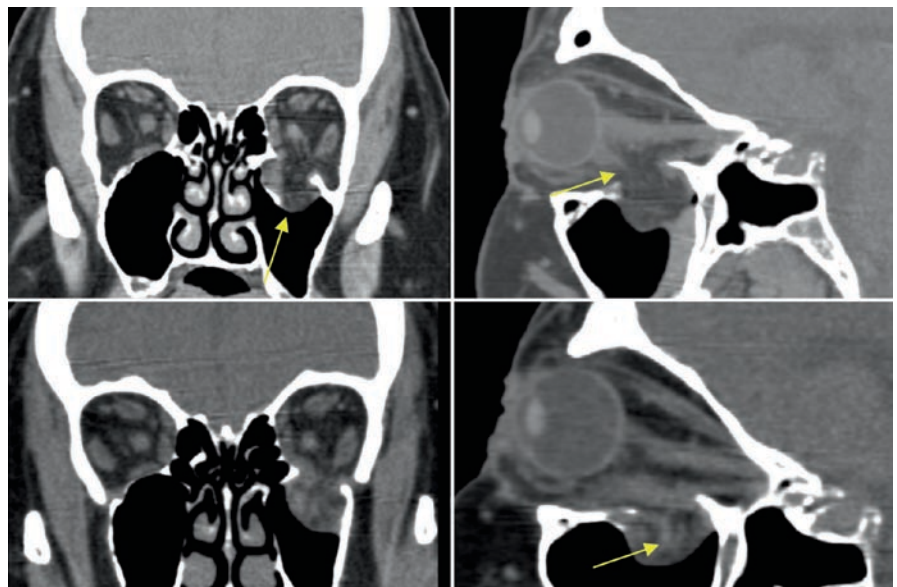


Fig. 6.33 Blow-out fracture of the orbital floor with massive herniation of orbital fatty tissue into the maxillary sinus (*arrow*) and with disruption of the inferior rectus muscle. *Upper row:* situation after the trauma. *Lower row:* scar formation (*arrow*) 3 months later

Fig. 6.34 Combined left zygoma and orbital floor fracture. (a) Clinical view: enophthalmus, disturbance of eye motility, limitation of down gaze with diplopia. (b) CT: downward displacement of orbital floor and orbital soft tissue including rectus inferior muscle (arrow) into the maxillary sinus

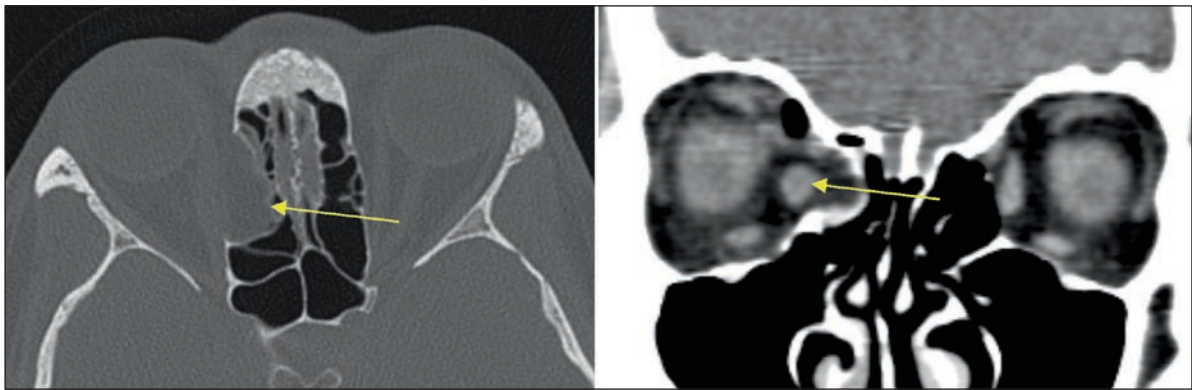
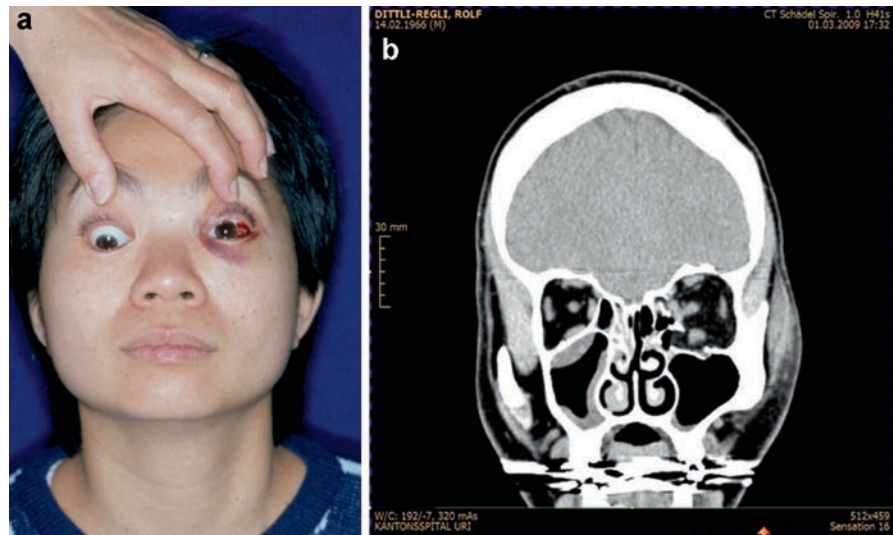


Fig. 6.35 CT images of blow-out fracture of the right medial orbital wall with displacement of the bone fragment together with fatty orbital tissue into the ethmoid (arrow)

6.3.3.2 Medial Orbital Wall Fractures

Dislocation of the paper-thin lamina papyracea ossis ethmoidalis results in a medial fatty tissue prolapse into the ethmoid cells and orbital enlargement, leading to an enophthalmus. Medial orbital wall fractures often result in disturbed horizontal eye motility (abduction) and a retraction syndrome (Figs. 6.35–6.37).

Characteristics of medial orbital wall fractures (blow-out fracture)

- Restricted horizontal movement
- Restricted abduction
- Globe retraction
- Subcutaneous emphysema
- Nasal hemorrhage
- X-ray: bone displacement/shading of the ethmoidal sinuses (20-70% in combination with orbital-floor fractures)

6.3.3.3 Orbital Facial Fractures, Types: Blow or Roof Fractures

Orbital roof fractures occur in approximately 5% of craniofacial fractures and may result in restricted eye motility, depressed position of the globe, ptosis (N.III) and neurological deficits (Klug and Machtens 1977; Ernst et al. 2004).

Clinical characteristics of orbital roof fractures

- Restricted elevation (rectus superior muscle)
- Ptosis (N. III)
- Depression of the globe (blow-in fractures)
- Neurological deficits (N.III)
- 5% of facial-fracture types: blow-out/blow-in

In the case of orbital roof impression, one can occasionally palpate flattening in the area of the

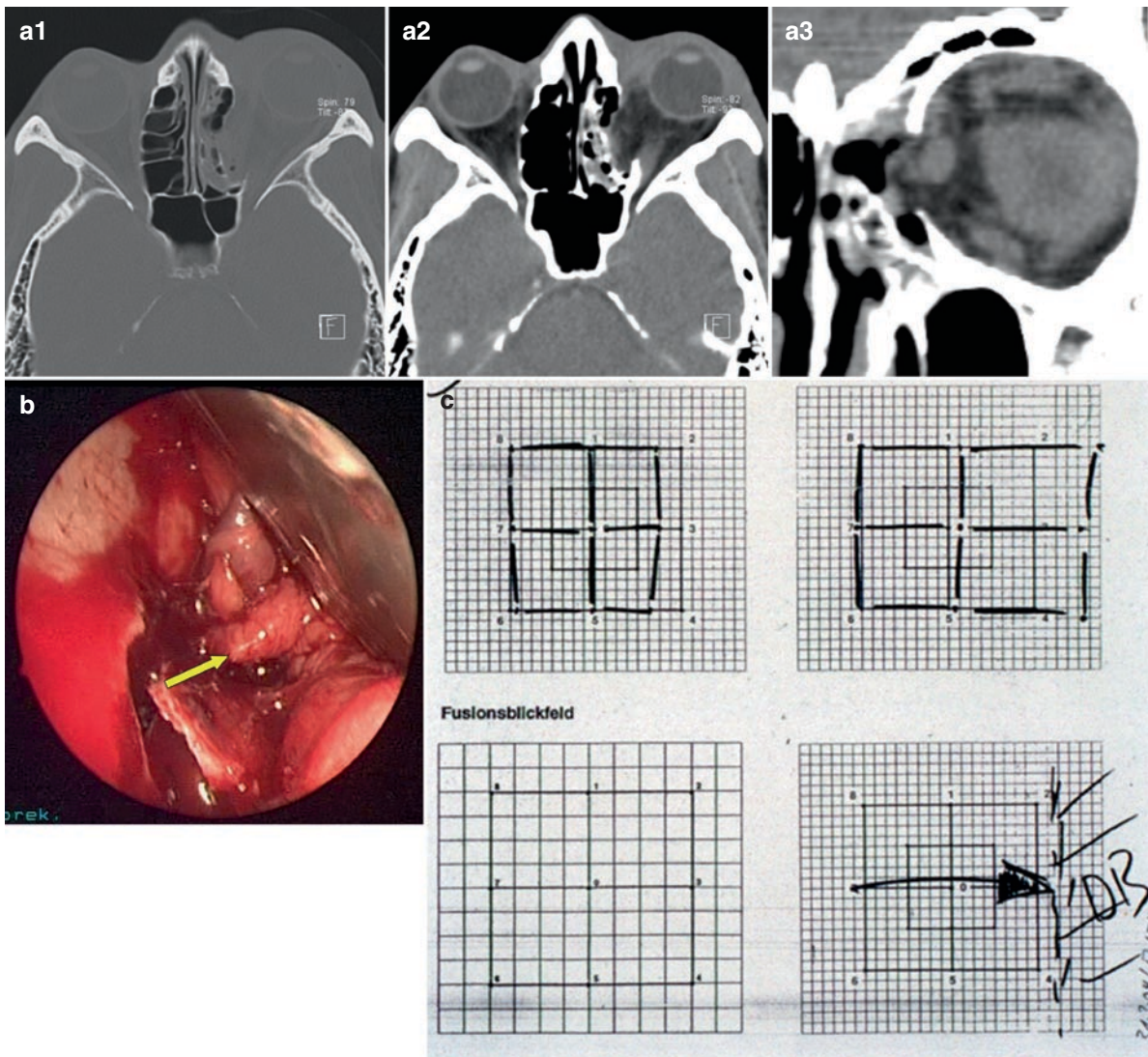


Fig. 6.36 Displaced fracture of the medial orbital wall with diplopia. (a) Transverse and coronal CT images, demonstrating displacement of the left medial orbital wall into the posterior ethmoidal cells together with a substantial amount of orbital fat.

(b) Transnasal endoscopic view: demonstration of fat tissue prolapses into the ethmoid (arrow). (c) Hess-Weiss test: restricted abduction with diplopia.

supra-orbital region. Extensive injuries with dural tears may lead to liquor penetration into the upper-lid, resulting in a palpable cushioned swelling or pulsation of the periorbital region.

Ptosis of the upper lid caused by direct or indirect damage to the levator palpebrae muscle may be present. Diplopia is often a result of hemorrhage or entrapment of the rectus superior muscle or direct damage to the supplying nerve (N.III). Hypesthesia of the fore-

head region occurs as a result of injury to the supraorbital nerve.

Surgical revision is indicated if the globe is displaced, if roof fragments are displaced cranially, and especially if there is additional rhinoliquorrhea and neuromuscular disturbance. Bone fragments of the orbital roof have to be reduced or removed carefully. Larger orbital roof defects should be closed with autogenous grafts or resorbable membranes (Figs. 6.38 and 6.39).

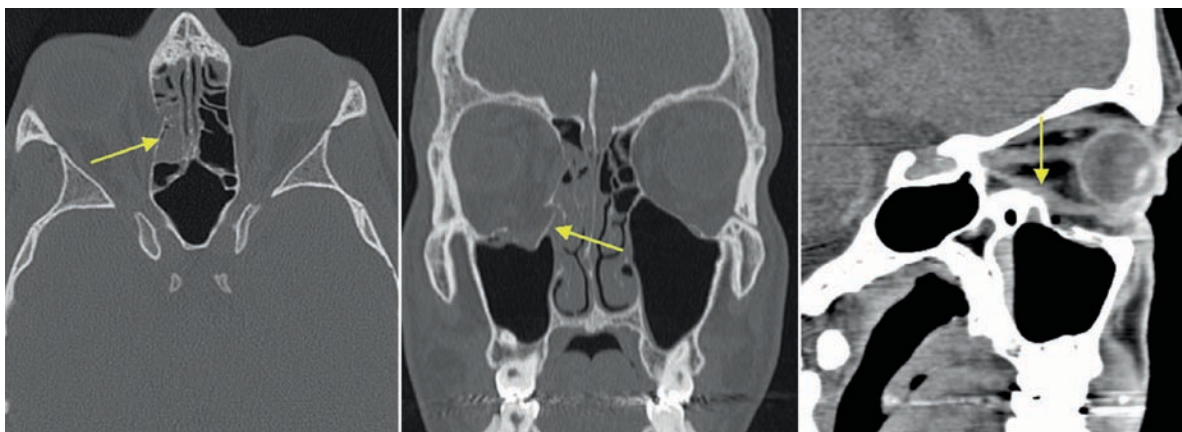


Fig. 6.37 Combination of medial and inferior blow-out fracture of the orbit. Only slight displacement of the medial orbital wall into the ethmoid and moderate displacement of the medial part of the

inferior orbital wall into the maxillary sinus (*arrow*). The sagittal CT image demonstrates swelling and displacement of the inferior rectus muscle over the edge of the orbital floor fracture (*arrow*)

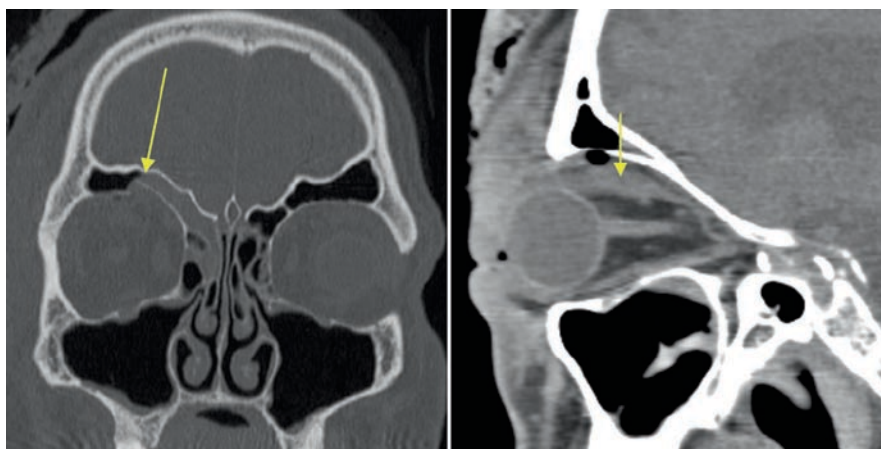


Fig. 6.38 Rare situation of a blow-out fracture of the orbital roof. Displacement of the fragment into the right frontal sinus (*arrow*). Hematoma along the superior rectus muscle and within the frontal sinus (*arrow*)

6.3.3.4 Multiple Wall Fractures

In complex facial trauma, multiple orbital wall fractures may lead to severely disturbed eye motility, neurological syndromes (e.g., OAS), and optical nerve injuries.

In orbital roof fractures, additional liquorrhea, pneumocephalus, or epidural hemorrhage is often found (Fig. 6.40).

Additional extraorbital injuries in orbital roof fractures

- Epidural hematoma
- Pneumocephalus
- Liquorrhea
- Fatty tissue prolapse into the frontal sinus

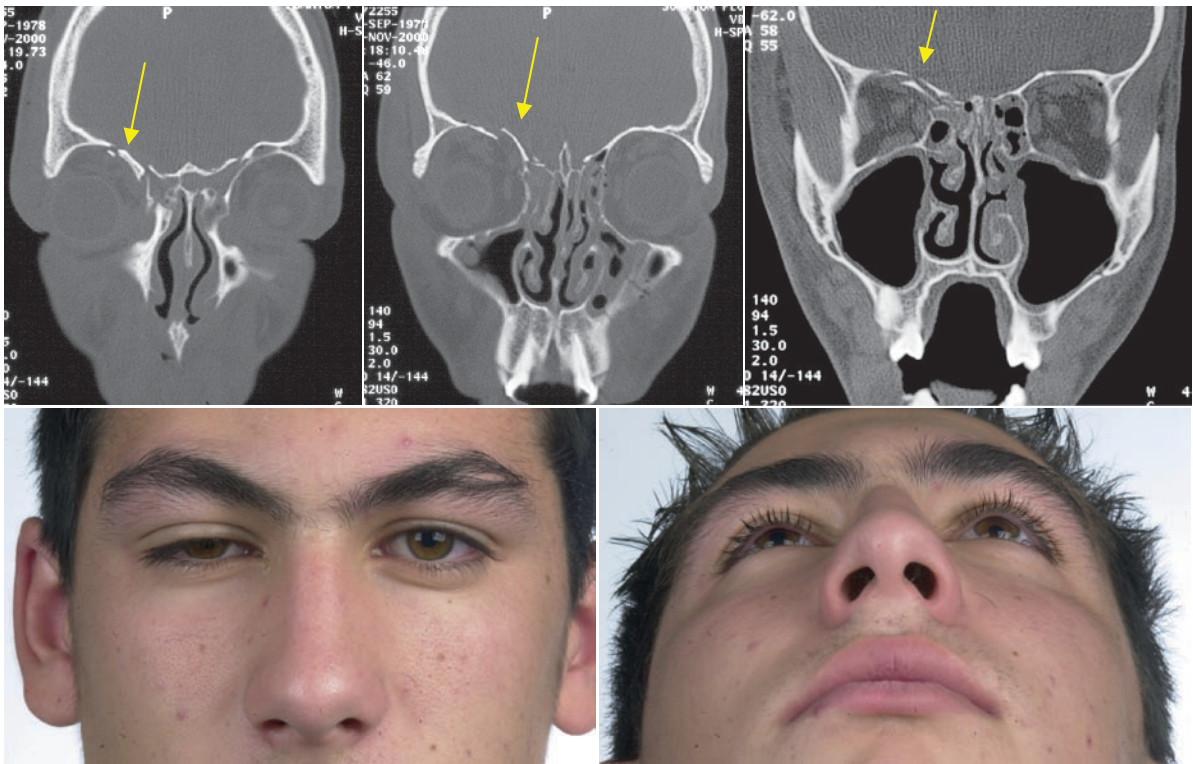


Fig. 6.39 Upper row: Comminuted fracture of orbital roof, sphenoid wing and cribriform plate (arrow) with consecutive disturbed motility (restriction: superior oblique and rectus muscles).

Lower row: Clinical presentation: enophthalmus, ptosis, and depression of the globe

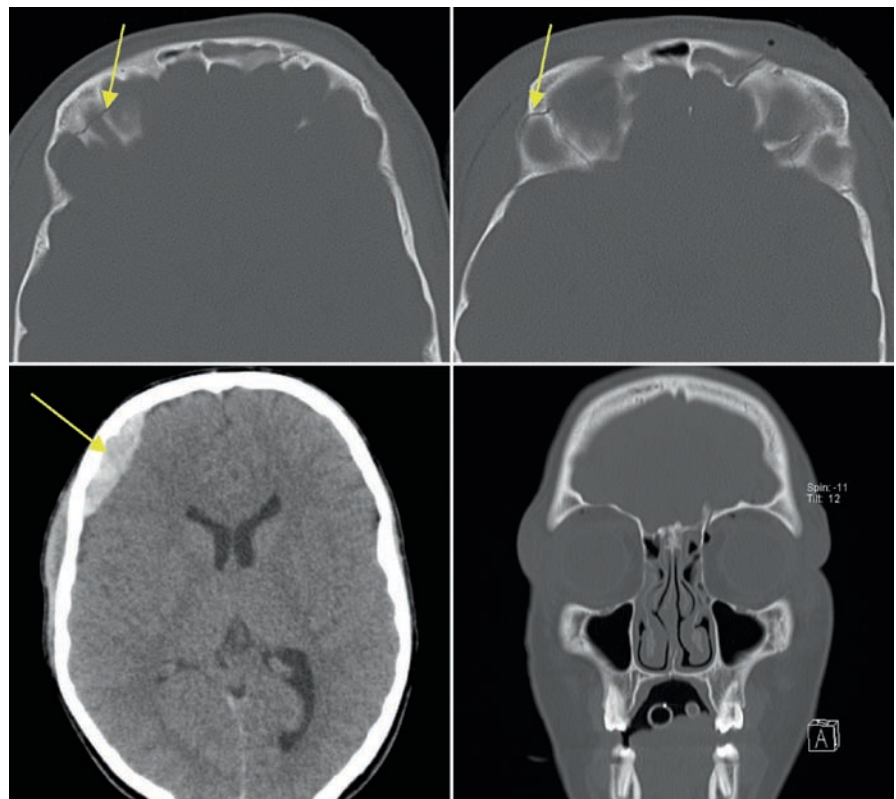


Fig. 6.40 Epidural hematoma after orbital roof fracture (arrows)

6.3.4 Fracture Signs

6.3.4.1 Clinical Manifestations

Clinical signs of orbital wall fractures are emphysema, orbital hematoma, infraorbital nerve hypesthesia, altered position of the globe, disturbed eye motility, retraction syndrome and changes of the palpebral fissure (Neubauer 1987; Stassen et al. 2003; Dutton and Al Qurainy 1991).

Clinical manifestations in orbital wall fractures

- Change in globe position
- Disturbance of eye motility
- Pseudoptosis
- Orbital emphysema
- Hypesthesia of the infraorbital nerve
- Retraction syndrome
- Change in width of the palpebral fissure

6.3.4.2 Change in Globe Position

The position of the globe is determined by the volume of the orbital fatty tissue, the periocular muscle balance, tissue hydration and tension of the ligaments and septa (Manson et al. 1980).

Factors influencing the globe position

- Volume of periorbital fatty tissue
- Balance of ocular muscle tone
- Ligament integrity
- Atmospheric pressure
- Tissue hydration

Changes in globe position are either due to an increase/decrease in osseous orbital volume or increase/decrease in orbital/periorbital soft-tissue volume. An increase in orbital volume/deficit in periorbital tissues results in an enophthalmus, muscular imbalance and disturbed eye motility; a reduction in bony orbital volume/increase in periorbital soft tissue will result in exophthalmus, disturbed motility and change in the globe position (Parsons and Mathog 1988; Stassen et al 2003).

6.3.4.3 Enophthalmus

An anterior-posterior change in the globe position, an enophthalmus is caused by either a fracture-related size increase of the orbital cavity or a reduction in soft-tissue volume (Manson et al. 1986; Stassen et al 2003).

Enophthalmus

Reasons for a relative or absolute increase in orbital volume with changes in the position of the globe (anterior-posterior)

Increase in orbital volume	Loss of support by orbital walls – fractures
Reduction in fat volume	Relative loss – herniation
Loss of fatty tissue	Irreversible loss – necrosis, fat liquefaction, fibrosis

6.3.4.4 Exophthalmus

An exophthalmus results from an inward displacement of bony wall fragments or through a pathological increase in orbital soft-tissue volume.

Exophthalmus

Reasons for a relative or absolute volume reduction with changes in the position of the globe (anterior-posterior)

- Inward dislocation of orbital wall fragments (blow-in fracture)
- Reduction of osseous orbital volume
- Retrobulbar – intraconal hematoma
- Edema – emphysema
- Subperiosteal hematoma

6.3.4.5 Vertical Displacement of the Globe

Vertical changes in the position of the globe are based on either a dislocated orbital floor fracture with antral soft-tissue prolapse or on an orbital roof fracture of the blow-in type.

Reasons for vertical globe displacement

Orbital floor fracture	Globe elevation – hematoma Globe depression – floor defect
Orbital roof fracture	Globe depression Hematoma, emphysema Fragment displacement (blow-in fracture)

6.3.4.6 Horizontal Changes in the Position of the Globe

Horizontal changes of the globe position originate from an inwards displacement of either the medial or lateral orbital walls, or are due to direct ligamental injury or avulsion of the insertion point (Rowe and Williams 1985).

6.3.4.7 Retraction Syndrome

In fractures with muscle entrapment or fragment piercing into the ocular muscles an additional retraction syndrome develops, during elevation (orbital floor fractures), ab-, and adduction (orbital wall fractures) of the globe.

6.3.4.8 Disturbances of Eye Motility

Traumatic disturbances of eye motility can be expected in 10-20% of midface fractures and in 47% of dislocated orbital wall fractures, and they are due to either mechanical or neurogenic reasons (Reny and Stricker 1973; Flick 1976; Lee 1983; Neubauer 1987; Dutton and Al Qurainy 1991).

Mechanical restrictions of eye motility result either from displacement of the globe or damage to the musculoskeletal system (Hardt and Sgier 1991; Joos U 1995; Dutton and Al Qurainy 1991).

Causes are muscle incarceration in the fracture gap, ligament entrapment, eye muscle injury, intraorbital or intramuscular hemorrhage, displacement of muscle insertion as well as secondary scarring and adhesions between muscle sheath and fracture margins.

Neurogenic disturbances evolve from traumatic damage to the cranial oculomotor nerves III, IV, VI, or damage to central nervous regions (Flick 1976; Hasso et al. 1979; Hardt and Steinhäuser 1979; Ghobrial et al. 1986; Dutton and Al Qurainy 1991).

Reasons for mechanical/neurogenic disturbances of the eye motility

- Muscle incarceration
- Ligament entrapment
- Injury to ocular muscles
- Intraorbital/intramuscular hemorrhage
- Nerve injuries (N. III, IV, VI)
- Displacement of ocular muscle insertion
- Scarring

A duction test and electromyography (EMG) may be used to differentiate between mechanical and neuromuscular eye motility disturbance (Flick 1976).

Differentiation of eye motility disturbance

	Duction test	EMG
Permanent mechanical oculomotor defect	Pathological	Normal
Temporary mechanical oculomotor defect	Normal	Normal
Neurogenic motility disturbance	Normal	Pathological

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7.1 Intracranial Injuries

7.1.1 Statistics: Intracranial Injuries in CF Fractures

Depending on the size and extent of the osseous injuries to the craniofacial area, there are associated intracranial injuries in about 20–30% of the cases (Manson et al. 1987; Neidhardt 2002). In the present series, 56% of all patients were found to have at least a dural tear, whereas in only 17% a dural injury was suspected before surgery. On imaging studies, additional cerebral contusions were seen in 42%, brain oedema in 11% and intracranial hemorrhages in 25%.

Frequency of intracranial injuries in craniofacial fractures (Neidhardt 2002/MKG-Luzern)

Contusion hemorrhages	42%
Pneumocranium	27%
Intracranial hematomas	25%
Cerebral edema	25%
Liquorrhea	18%

7.1.2 Principles of Neurotraumatology (Rengachary and Ellenbogen 2004)

The basic management principles for patients with *traumatic brain injury* (TBI) apply equally to those who happen to sustain a craniofacial injury.

Head injuries are probably the most common condition neurosurgeons have to deal with. Even though

it is commonly believed that mortality from head injury has significantly declined over the past five decades due to a major improvement in immediate medical care, TBI continues to be a major cause of mortality and morbidity, particularly in the young and it will remain so in the foreseeable future.

The treatment goal is orientated in two directions. Firstly, to repair the immediate effects of the primary injury and facilitate healing. This is mostly achieved with surgical intervention. Secondly, to prevent the development or at least minimize the effects of secondary injury.

This is attempted with medical treatment, such as adequate oxygenation and maintenance of cardiovascular, cerebrovascular, fluid, and electrolyte homeostasis. Depending upon circumstances, this also requires surgical intervention, such as drainage of cerebrospinal fluid (CSF) or decompressive craniectomy.

- *Classification of head injuries*

Neurosurgeons and neuroscientists use different aspects of TBI for its classification:

Severity of injury, mechanism of injury, morphology with focal or diffuse lesions, as well as course of time in primary and secondary injuries

- *Severity*

Since its inception in 1974, the Glasgow Coma Scale (GCS) has gained worldwide acceptance in describing the level of consciousness of patients after trauma and in nontraumatic conditions affecting consciousness (Tables 7.1 and 7.2).

The scale is simple, language-, and culture-independent and provides a high interobserver reliability. It is used to categorize the severity of head injury into three groups (Teasdale and Jennett 1974).

¹Contributed by Karl Kothbauer, Kantonsspital Luzern (CH), Neurosurgical Department, Luzern, Switzerland

Mild head injury	GCS 15–14
Moderate head injury	GCS 13–9
Severe head injury	GCS 8–3

Only patients with severe head injuries are unconscious (i.e., GCS 8 or smaller).

- Mechanism

Closed head injuries are all injuries without an open communication between the outside and the brain and/or CSF spaces. Stab and gunshot wounds are referred to as penetrating injuries. Obviously overlap and combinations exist. The majority of craniofacial injuries therefore fall into the category of open head injury.

- Morphology

Initial imaging studies enable structural brain injuries to be anatomically distinguished between diffuse and

focal brain injuries. Again overlap exists, as every TBI, however mild, with a history of brief loss of consciousness and a memory gap is by definition a mild type of diffuse brain injury and usually one that shows no focal signs of injury on computed tomography (CT) imaging. Extracerebral hematomas, circumscribed brain contusions, traumatic subarachnoid hemorrhage, skull, or skull base fractures are focal injuries that may occur in any severity category as defined by GCS.

- Time course

The initial physical impact causes a primary injury which may affect the brain and soft tissues with lacerations, fractures, contusions, etc.

A massive wave of secondary injuries may occur in patients with severe head injuries in the days following the initial trauma. This may result in elevated intracranial pressure (ICP) and consequently in relative impairment of brain tissue oxygenation and disruption of cerebrovascular autoregulation. The secondary injury forms the basis of all surgical and intensive care measures used for patients with severe head injuries.

- Patient assessment

As far as possible, a neurological examination is required for each patient with TBI, in addition to assessing the GCS. This includes pupillary size, symmetry, and response to light, eye movements, oculocephalic testing, motor power and testing of sensory function. Certainly note must be taken of additional injuries, such as spinal injuries, thorax, and abdominal injuries, peripheral fractures and craniofacial injuries.

- Diagnostic imaging

Cranial CT (CCT) is the modality of choice to evaluate all cranial injuries. For the neurosurgeon, the standard X-rays have become increasingly irrelevant in the light of excellent high-resolution and readily available CCT. Magnetic resonance imaging (MRI) may reveal additional information that may not be apparent on CCT, such as multiple small lesions in patients with normal CT scans. However, at present, MRI is not routinely used for head-injury patients. Other modalities such as cerebral angiography are used in rare cases of vascular injury at the skull base or after penetrating head injuries.

Table 7.1 Glasgow Coma Scale

Spontaneous	4
<i>Eye opening</i>	
To call	3
To pain	2
None	1
Obeys commands	6
Localizes pain	5
<i>Best motor response</i>	
Withdrawal	4
Abnormal flexion	3
Extension	2
None	1
Oriented	5
Confused	4
<i>Verbal response</i>	
Inappropriate words	3
Incomprehensible	2
None	1

Table 7.2 Glasgow Outcome Scale

Good recovery (G)	Patient returns to pre-injury level of function
Moderately disabled (MD)	Patient has neurologic deficits but is able to look after himself
Severely disabled (SD)	Patient is unable to look after himself
Vegetative (V)	No evidence of higher mental function
Dead (D)	

7.1.3 Management of Mild Head Injuries (GCS 14-15)

Mild head injuries make up 80% of all head injuries, e.g., traumas with brief loss of consciousness and amnesia.

- Diagnostic workup (Stiell et al. 2001)

Since only about 3% of all patients with mild head injuries are believed to deteriorate due to an intracranial lesion, which develops in the hours following the initial injury, there is considerable controversy about the pros and cons of optimal versus most cost-effective management. Risk factors from delayed deterioration have been established in various efforts to clarify this issue and put it beyond personal or institutional “policy”. These risk factors include: ambiguous accident history, high-impact trauma (e.g., man vs truck), retrograde amnesia over 30 min duration, continued GCS < 15 2 h after injury, clinical, or radiographic evidence of skull fractures, or skull base fractures, age over 60–65 or under 2 years, coagulation disorders or medical anticoagulation with vitamin K antagonists or treatment with platelet inhibitors.

There are no guidelines as to whether or not blood tests should be obtained routinely. It is probably advisable, since the patients are being checked for a possible lesion which may require emergency surgical intervention.

These risk factors characterize victims with mild head injuries who should be referred to CT scanning.

- Intervention

With or without CT scan, it is advisable to keep the patient in hospital for 24 h for neurological and medical observation. There are no strict management guidelines and without doubt in many institutions patients are discharged into the care of their families when a CT scan has documented the absence of an intracranial mass lesion.

Decrease in GCS by two points must prompt a CCT or a repeat scan if the former was normal. The presence of a mass lesion may then require surgery. If this is not the case it may be possible to discharge such a patient after approximately 24 h, similar to patients with no clinical deterioration.

- Rehabilitation

Most patients with mild head injury will not require additional care 2 weeks post injury. Protracted headaches

and mild disturbances in memory regeneration and concentration may occur.

7.1.4 Management of Moderate Head Injuries (GCS 13-9)

About 10% of patients with TBI suffer from mild head injuries. About 10% of this group will deteriorate and lose consciousness shortly following trauma.

- Diagnostic workup

CCT is indicated for all patients in this group. An abnormal scan should be repeated after 24 h even if no surgical intervention is necessary. All patients must also have definitive imaging of the cervical spine, nowadays preferably a spiral CT, which provides optimal imaging and avoids tiring discussions about incomplete visualization of the cervicothoracic section. These patients should have a routine preoperative laboratory workup.

- Intervention

Patients with moderate head injuries are monitored in the same way as those with mild head injuries. However, it is unlikely that discharge is possible after 24 h. Even if no deterioration or further problems occur, such patients are likely to remain hospitalized for a minimum of 2–4 days.

- Rehabilitation

A significant number of patients with moderate head injuries will require at least some form of rehabilitation. This may be on an inpatient or outpatient basis.

7.1.5 Management of Severe Head Injuries (GCS 8-3)

Patients with severe head injuries, unable to follow commands, require urgent and persistent attention. Cardiorespiratory stabilization is the first priority for these patients. Hypotension <90 mmHg of systolic

blood pressure is associated with subsequent ICP elevation and poor outcome. Patients in this category must be intubated early.

- Diagnostic workup

Cranial CT and CT of the cervical spine are obligatory studies. Other imaging studies must be done as appropriate since additional severe injuries are frequent, such as long-bone or pelvic fractures (30%), craniofacial injuries (20%), chest injuries (20%) or intrathoracic and intraabdominal injuries.

The neurologic examination beyond assessment of the GCS may be limited to pupillary size and response to light, blink reflex and oculocephalic maneuvers, as well as caloric vestibular testing. Bilaterally dilated and fixed pupils indicate severe transtentorial herniation and inadequate brain perfusion.

Additional imaging studies, such as MRI, may be indicated for associated spinal cord injury. Cerebral angiography may at times be important in case of severe orificial hemorrhage, which may only be controlled by endovascular techniques.

Common findings on CCT are intracranial mass lesions, such as epidural and subdural hematomas. These require craniotomy and evacuation in most instances (see below). Furthermore, brain contusions may be seen as hypodense areas or hyperdense in case of hemorrhagic contusion.

Traumatic subarachnoid hemorrhage is a frequent finding and indicates added severity of the initial injury but does not carry similar significance in and of itself as subarachnoid hemorrhage from spontaneous rupture of a cerebral aneurysm. Air inclusion indicates open fracture or skull base fracture. Today, fractures are well visualized with high-resolution spiral CT scanners.

Further findings on CT scans may be ventricular hemorrhage or diffuse cerebral edema. Hemorrhagic contusions are usually located at the base of the frontal and temporal lobes, wherever the cortex borders onto rugged bone edges of the skull base. So-called “contrecoup” contusions are frequently seen opposite to the impact site.

- Management of severe head injuries (Markwalder and Seiler 1985)

At times, an intubated and sedated patient delivered to a trauma centre with a presumed low initial GCS may have a normal CCT. Under certain circumstances it

may be in order to wait for such a patient to awaken or antagonize medication in order to fully assess the clinical situation. Many patients may, in fact, have higher GCS scores and the initial low scores may be the result of poor reporting or simply hemodynamic instability at the scene of the accident.

In all other circumstances, patients with severe head injuries must be monitored in the setting of an intensive care unit. An ICP monitoring probe should be placed, together with a ventricular drain. The latter provides direct hydrostatic ICP measurement and access to release CSF as a measure to reduce elevated ICP. Further monitoring probes may be placed in the cerebral parenchyma as appropriate. Differential measurements are often necessary to monitor brain temperature, tissue pO_2 , microdialysis, and jugular pO_2 . The comatose and, in most cases, heavily sedated patient cannot usually be assessed clinically and thus the physiological parameters measured with these devices, combined with hemodynamic parameters obtained from invasive measurements, substitute the direct monitoring.

All patients should have a second CCT within no more than 12 h of injury or if ICP rises above 25 mm Hg. This may indicate a secondary space occupying hemorrhage, which may require surgery. In the absence of a surgical lesion increasing ICP must be treated in conjunction with the cerebral perfusion pressure (CPP). Currently the recommended CPP is 60–65 mm Hg. The mean arterial pressure (MAP) may have to be adjusted with volume or vasopressors. CPP results from the MAP minus the ICP.

Intervention to keep ICP within normal limits should be initiated when ICP consistently exceeds 20–25 mm Hg.

There are numerous *strategies* utilized to lower ICP and therefore to preserve CPP to avoid secondary ischemic brain damage. Positioning the patient with the head and upper body elevated by 15–30° enables free venous outflow and thus helps keep ICP and cerebral circulation within normal limits. Sedation, preferably with a short-acting substance such as propofol reduces the body's response to stress and blunts vegetative irritation, it also limits the brain's oxygen requirements and thus helps prevent elevated ICP. Adequate pain medication with fentanyl or remifentanyl minimizes pain induced stress, vegetative arousal and subsequent hypertension.

Mannitol is very effective in reducing elevated ICP but has the disadvantage of accumulating in contused areas of the brain where the blood brain barrier is disrupted. Thus, it may actually worsen the local swelling around contusions. Therefore, it should only be used in situations of critical ICP elevation; for instance, on the way to surgery with a newly detected hematoma. Long-term use in regular intervals, such as 2–4 h, should be discouraged.

Other diuretics, such as furosemide and acetazolamide, are effective in reducing CSF production and ICP in the absence of trauma. They are, however, rarely used in the setting of severe head injury, due to their limited efficacy in the TBI setting.

Hyperventilation is a very effective short-term intervention to lower elevated ICP. The physiologic response is due to cerebral vasoconstriction, which in turn reduces cerebral blood flow (CBF) and CBV and consequently ICP. It has been shown however, that sustained hyperventilation lowers the paCO_2 to around 30 mm Hg or less. This increases the incidence of significant focal cerebral ischemia and, thus, has a net deleterious effect and a negative effect on outcome. Hyperventilation is, therefore, only used in critical situations (see above, like mannitol) or under the guidance of continuous measurement of global oxygen extraction with jugular pO_2 assessment and, if available, with focal tissue pO_2 measurement.

The use of steroids in TBI has been abandoned since the CRASH study proved the deleterious effects of steroids on long-term TBI outcome.

Barbiturates are very effective in reducing cerebral oxygen requirements, CBF, and thus ICP. However, the side effects of shutting down the airway clearance mechanisms, immune response dysfunction, hypotension, and subsequent requirement for increased need of vasopressors often make this treatment problematic. It should only be used in acute situations where other measures are not effective in controlling ICP and preserving CPP. The use of barbiturates is often discussed even after performing a craniectomy to control ICP.

Anti-epileptic drugs are frequently used on a prophylactic basis, particularly in the United States. However, sedation is in itself prophylactic for seizures and seizures in the acute phase of severe head injury in no more than 5–15%. The use of standard anticonvulsants, such as phenytoin, is also problematic as more recent studies have shown that patients who initially received this drug demonstrated a reduced rehabilitation response at a later phase. We only used anticonvulsants in patients who had suffered seizures.

7.1.6 Neurosurgical Management of Intracranial Hematomas

The large majority of treatment recommendations for the management of traumatic intracranial mass lesions are experience-rather than evidence-based. No-one will ever undertake a prospective trial to prove that surgical evacuation of an epidural hematoma is superior

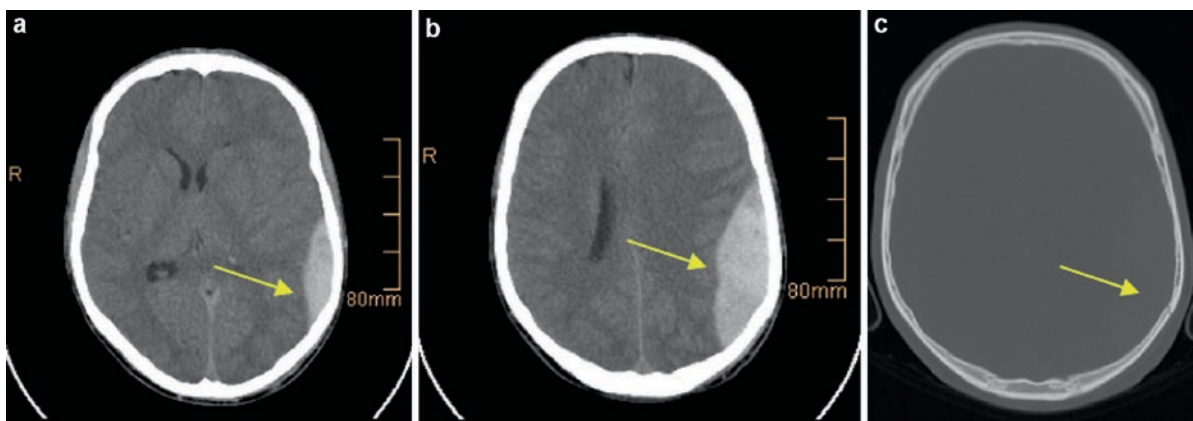


Fig. 7.1 (a, b) Epidural hematoma (EDH) (arrow) in a 24-year-old man after head trauma. (c) Skull fracture in the region of the hematoma (arrow). EDHs are the result of injury to an epidural artery by a skull fracture

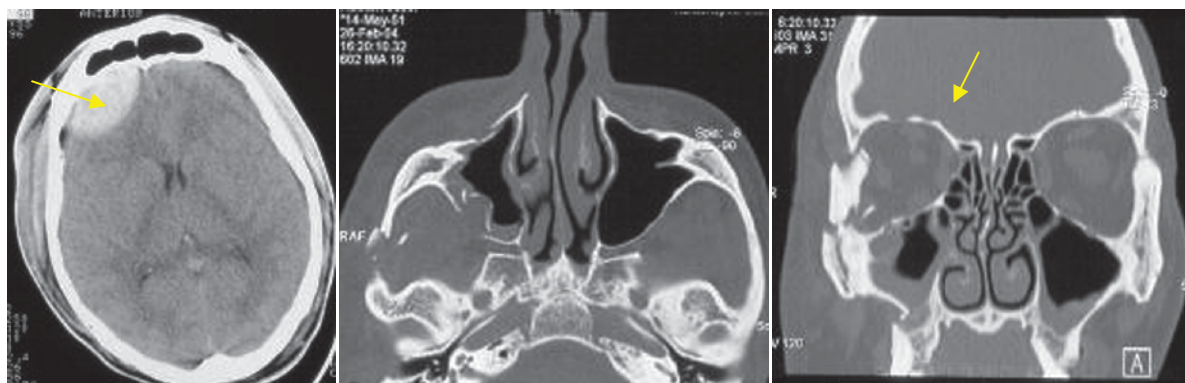


Fig. 7.2 Supraorbital epidural hematoma (*arrow*) associated with fracture of the orbital roof on the right (*arrow*) following complex fracture of the zygomatic bone

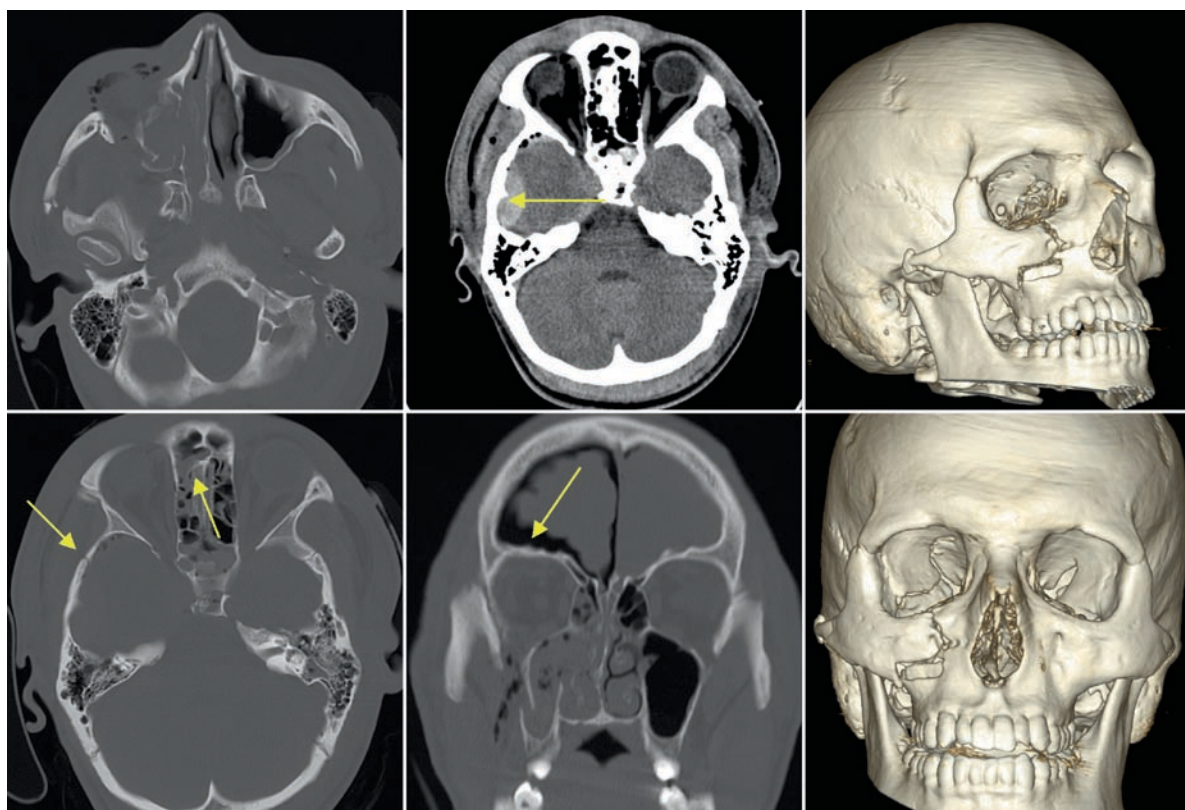


Fig. 7.3 Complex cranial trauma. Displaced lateral midface fracture with inward displacement of the zygomatic bone. Right temporal skull fracture with EDH (*arrow*). Frontobasal fracture with pneumatocele (*arrows*)

to conservative treatment! Nevertheless, the literature permits to formulate certain guidelines for the indications of surgery, use of adjuvant medical treatment and timing of intervention.

- Epidural hematoma (EDH) (Bullock et al 1996, 2006a)

EDHs are frequent in the younger age groups as the dura becomes increasingly adherent to the inner table with increasing age. Typically, the epidural clot evolves in the context of a skull fracture that crosses the course of a meningeal vessel, particularly the middle meningeal artery, which ruptures and bleeds. Patients with epidural hemorrhage are the classical “talk-and-die”

patients, as their initial injury may appear mild but their deterioration may be dramatic, and the course without intervention lethal.

EDHs are evacuated via a standard craniotomy centered on the location of the clot. Bleeding is controlled using bipolar coagulation and the dura must be secured to the skull using strong tack-up sutures.

Guidelines for surgical indications have recently been defined. Hematomas larger than 30 cm³ should be evacuated regardless of GCS and in a timely fashion. Nonsurgical management with serial imaging and close neurological monitoring can be used for smaller hematomas with thickness less than 15 mm *and* midline shift less than 5 mm *and* GCS greater than 8 *and* no focal neurological deficits (Figs. 7.1–7.3).

- Acute subdural hematoma (SDH) (Bullock et al. 1996, 2006b)

By and large the presence of an acute SDH indicates a more serious brain injury than the presence of an epidural clot. Direct brain injury with surface contusions is a frequent source of subdural bleeding, as is rupture of bridging veins as a result of traumatic shifting of the brain on impact.

Acute SDHs are usually evacuated via a large (“trauma flap”) craniotomy with bone flap removal

and a duroplasty to provide room for brain swelling. It is advised to place an ICP probe as well as a ventricular drainage catheter at the same time in all patients with a GCS of 8 or less.

The recently published guidelines recommend surgery for acute SDHs with a thickness of greater than 10 mm *or* a midline shift of greater than 5 mm regardless of GCS. Furthermore, patients with acute SDHs smaller than indicated above *but* with a decrease of at least 2 points of GCS between injury and hospital admission *and/or* pupillary asymmetry *and/or* an ICP exceeding 20 mm Hg should also undergo surgery as soon as possible (Figs. 7.4 and 7.5).

- Parenchymal contusions (Bullock et al. 1996, 2006c)

Surgical management of traumatic parenchymal lesions depends upon the assessment of tissue damage exerted by a focal contusion and the effect such a lesion has on the overall elevation of ICP. Due to the heterogeneity of parenchymal lesions, treatment recommendations vary. Parenchymal lesions causing progressive neurological deterioration, medically refractory ICP elevation or mass effect on CT should be surgically removed.

Patients with GCS 6–8, who have frontal or temporal contusions greater than 20 cm³ and midline shift of 5 mm or greater and cisternal compression, as well as any patients with lesions larger than 50 cm³, should undergo surgery.

If surgery is performed, in the majority of cases it will be a craniotomy with the removal of the contusional hemorrhage and meticulous hemostasis. Other treatment options, such as large hemispheric or bifrontal craniectomies or smaller focal decompressions, exist and their applicability depends upon the individual circumstances (Figs. 7.6–7.9).

- Depressed skull fractures (Bullock et al. 1996, 2006d)

The management of depressed skull fractures has varied over the past two decades, from an aggressive surgical to a more conservative approach. The recent guidelines again put more emphasis on surgery and state that all management options include antibiotic prophylaxis. Indications for surgery are open calvarial fractures depressed to a greater extent than the thickness of the cranium or the risk of infection. Infections are best combated with surgery. Conservative management is an option, if there is no evidence of dural penetration, significant intracranial hematoma, frontal



Fig. 7.4 Large SDH covering the right cerebral hemisphere (arrow)

Fig. 7.5 Large SDH with moderate compression of the right frontal lobe (*arrow*). The subdural space covers the brain surface and continues along the falx cerebri (*arrow*). Moderate compression of the frontal lobe by the surrounding hematoma. Hematosinus maxillaris indicating midface fracture

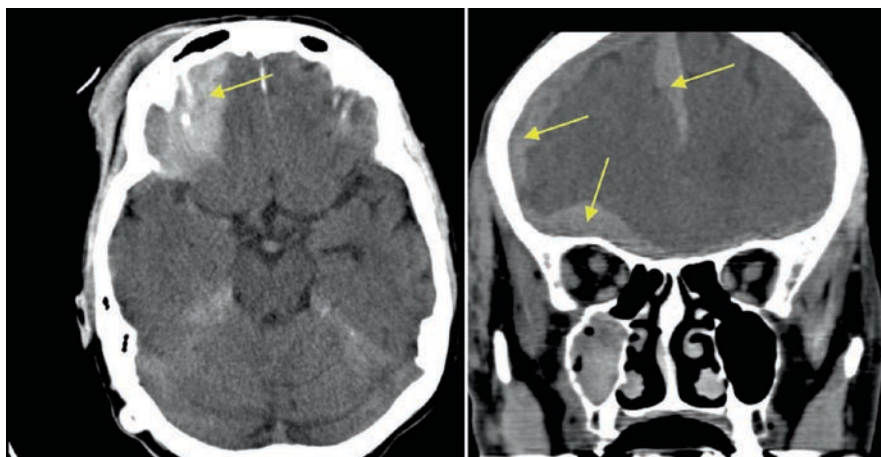
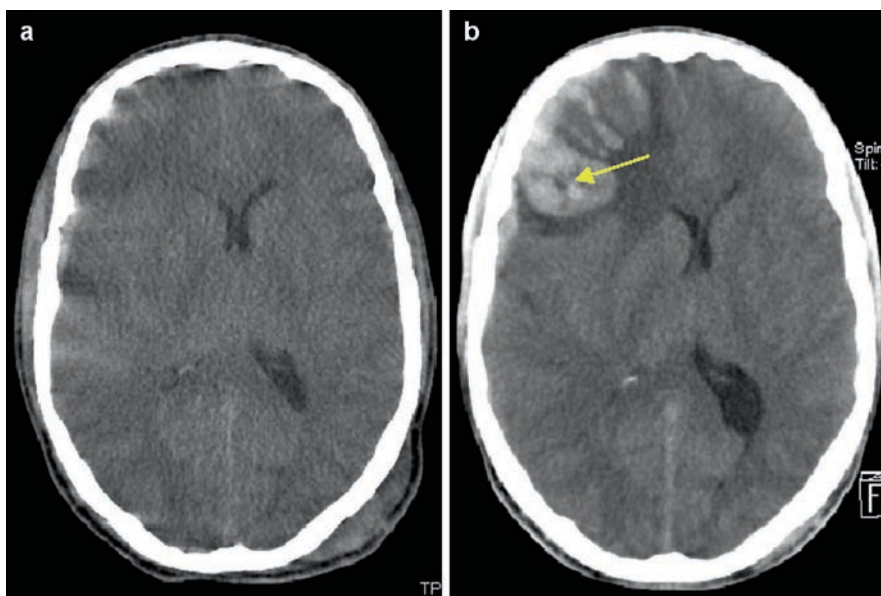


Fig. 7.6 Traumatic contusional hemorrhages in the right frontal lobe of a 21-year-old man. The initial CT on the day of injury shows little evidence of structural injury, only a small amount of SDH with no mass effect (**a**). Four days later, a significant hemorrhagic contusion (*arrow*) with surrounding edema has developed with a moderate mass effect (**b**) and led to significant clinical worsening



sinus involvement, infection, pneumocephalus, or gross wound contamination. In order to prevent infection surgery should be performed early (Fig. 7.10).

- Chronic SDH (Markwalder 1981; Markwalder and Sailer 1985)

Chronic SDHs frequently (but not exclusively) occur in the elderly and are significantly associated with oral anticoagulation, use of platelet inhibiting medication, other factors reducing normal hemostasis, and a recent history of, often only mild, head trauma. Not infrequently they start as small, nonsurgical acute or subacute hematomas and then undergo a process of evolution. This includes the accumulation

of fluid, the formation of neomembranes and thus an increase in volume, which may cause a mass effect and consequently a variety of symptoms, including headache, gait problems, ataxia, confusion, dysphasia, and hemiparesis. Once significant symptoms are present, surgical evacuation is recommended, although spontaneous remission sometimes occurs.

Surgical techniques vary from single bur-hole drainage to double bur-hole drainage to craniotomy. Most authors recommend draining the liquid during surgery and leaving a subdural drain for 24–48 h to complete drainage and subsequent re-expansion of the brain. Chronic SDHs are rare in the context of craniofacial injury (Fig. 7.11).

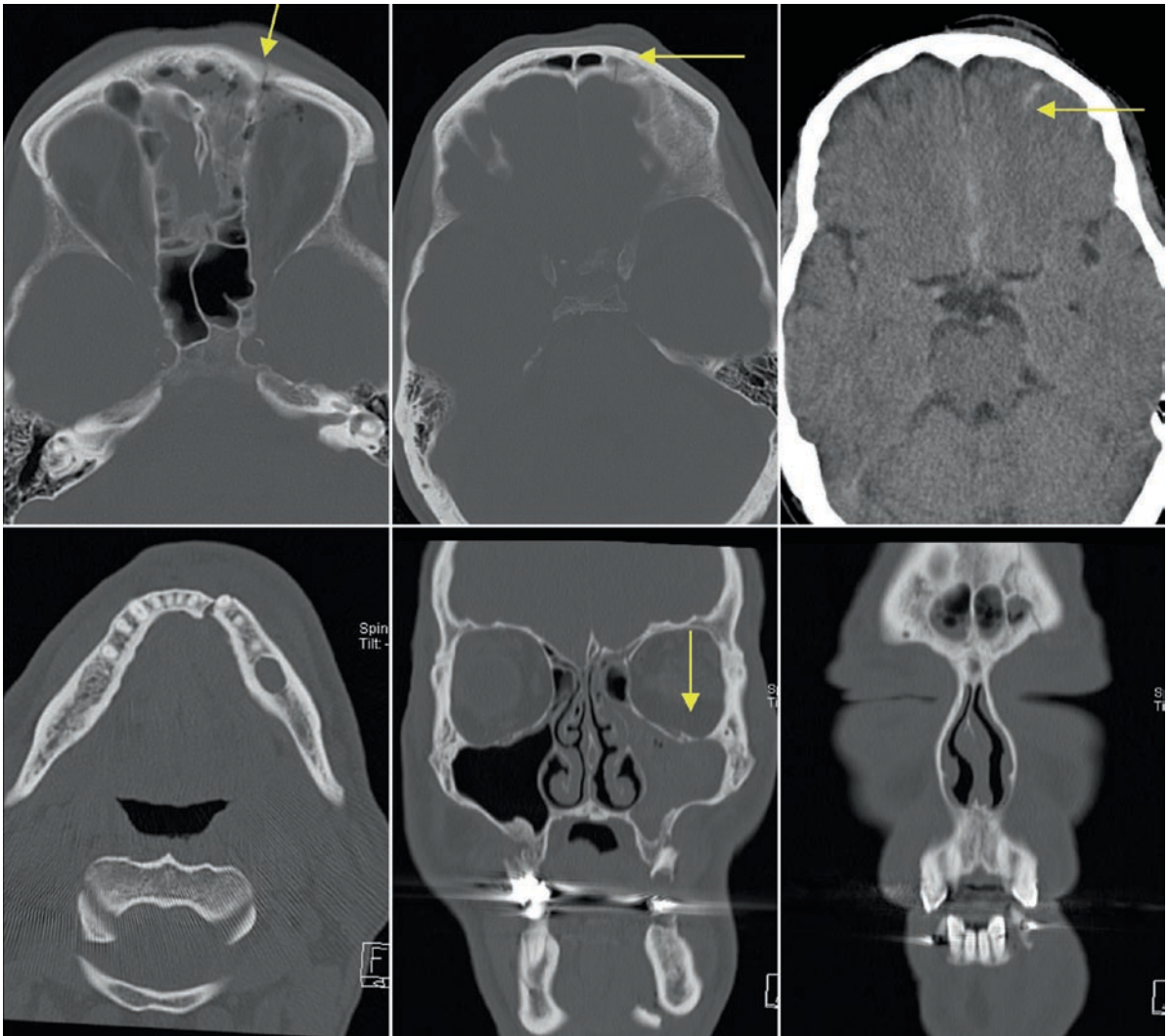


Fig. 7.7 Small contusion injuries to the left frontal lobe (*right upper image*) (*arrow*) after fronto-facial trauma with fracture of the mandible, left frontal bone, left orbital roof and left orbital floor (*arrow*)

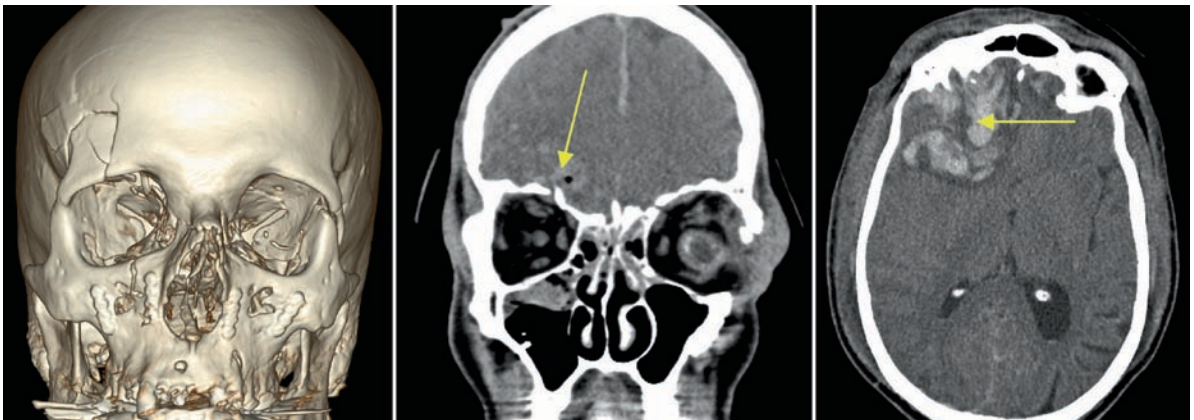


Fig. 7.8 Fronto-temporal skull fracture and frontal skull base fracture (*arrow*) with major contusion hemorrhages in the right frontal lobe (*arrow*). Healed midface fracture with osteosynthesis plates on both maxillae from a previous trauma

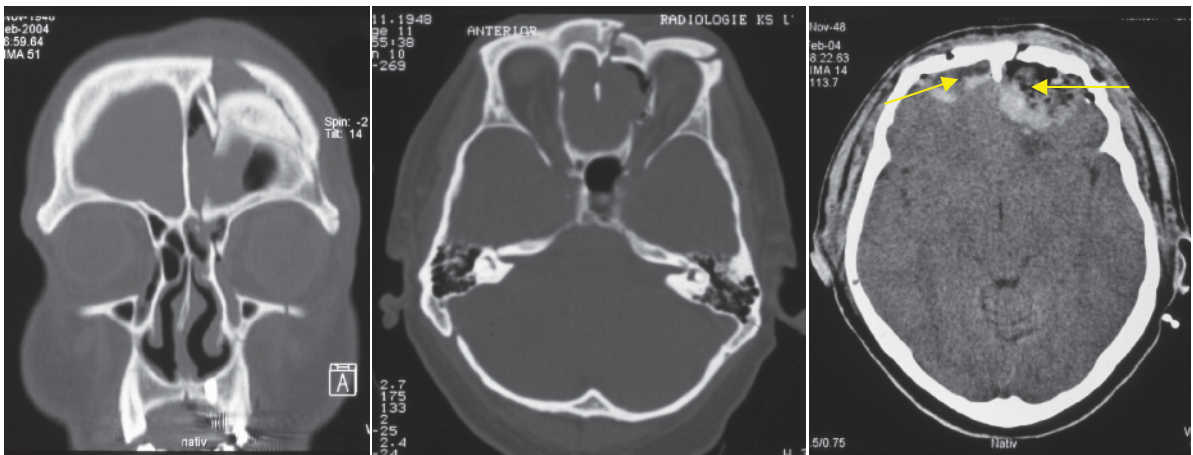


Fig. 7.9 Traumatic parenchymal hemorrhagic contusions in the frontal lobes (*arrow*) after a left cranio-orbito-facial fracture

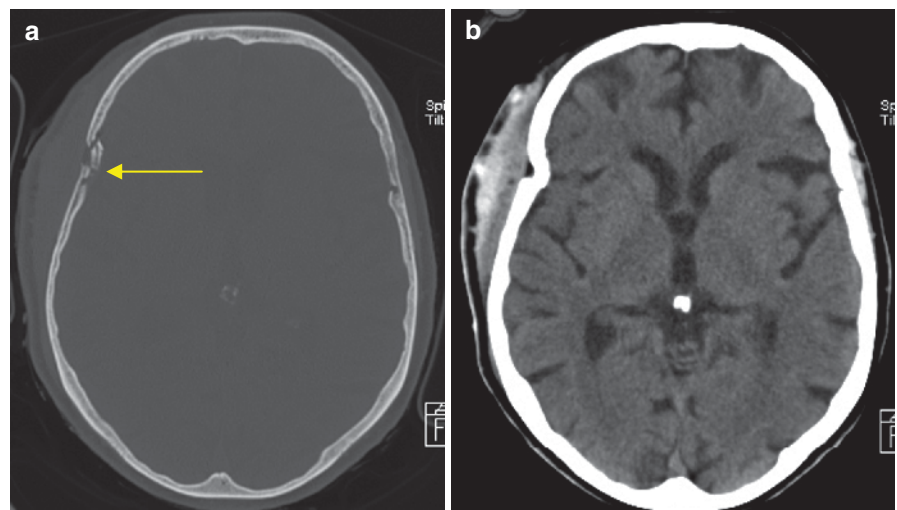


Fig. 7.10 Depressed skull fracture in an 84-year-old woman. There is no evidence for intracranial injury (a) but a clearly depressed small bone fragment (*arrow*) (b). Treatment was conservative with a short course of prophylactic antibiotics

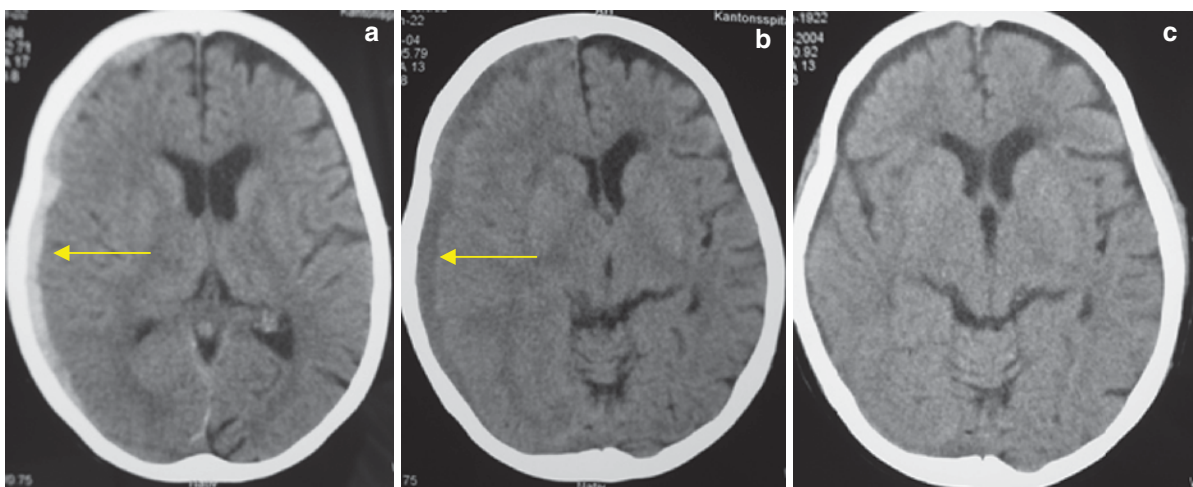


Fig. 7.11 Acute SDH (*arrow*) (a), converting into a chronic SDH (*arrow*) (b) with slow regression over months (c)

7.2 Management of Skull Base and Dural Injury

(Loew et al. 1984; O Brian and Reade 1984; Buchanan et al. 2004)

Traumatic CSF leakage occurs with skull base fractures adjacent to the pneumatized paranasal sinuses, the temporal bone and the mastoid, where the dura and presumably the arachnoid rupture.

Cerebral injury directly following impact and infection following bacterial migration from the pneumatized spaces may occur (Jamieson and Yelland 1973; Flanagan et al. 1980; Hubbard et al. 1985; Georgiade et al. 1987; Schmideck and Sweet 1988).

CSF leakage ceases without intervention within 24 h in 35% of cases, within 48 h in 68% and in 85% within a week (Mincy 1966; Schmideck and Sweet 1988). This has a major impact upon whether or not CSF leakage requires surgical repair.

Average ebbing time in manifested liquorrhea in frontobasal fractures without intervention

24 h	35%
48 h	68%
1 week	85%

(Schmideck and Sweet 1988)

7.2.1 Skullbase Fractures with CSF Leakage

Persistent rhinorrhea in the context of a frontal skull-base fracture requires surgical intervention (Fonseca and Walker 1991; Godbersen and K  gelgen 1998a, b).

Conservative management, particularly of multi-fragmented frontobasal fractures, is associated with a significant risk of meningitis (Loew et al. 1984).

Surgery decreases this risk. The rate of postoperative meningitis has been reported as low as 3.8% (McGee et al. 1970; Spetzler and Zabramski 1986; Sakas et al. 1998; K  stner et al. 1998). For this reason, early surgery is attempted in frontobasal fractures with rhinorrhea with and without CT evidence of intradural air. This principle is similarly applied if rhinorrhea and infection occur at a later stage after injury (Probst 1971; Russel and Cummings 1984; Probst 1986).

Controversy exists concerning the exact timing for the surgical repair of frontobasal dural defects (Dagi and George 1988; Schaller 2002).

The following scenarios are managed in different ways:

7.2.2 Skullbase Fractures with CSF Leak without Severe TBI

Isolated injuries including dural rupture are usually repaired late (Probst and Tomaschett 1990).

7.2.3 Skullbase Fractures with CSF Leak with Severe TBI

In the acute phase after severe TBI, brain edema, elevated ICP and the presence of cerebral contusions, the brain is vulnerable to further injury by surgical manipulation. Therefore, repair of a persistent CSF leak is usually recommended 1–2 weeks after the injury (Loew et al. 1984; Sprick 1988). Surgery should be delayed in the case of persistent impaired consciousness, particularly with DI or hyperthermia.

7.2.4 Combined Frontobasal-Maxillofacial Fractures with CSF Leakage with or without Severe TBI

In the context of a possible severe TBI, with increased complexity of the injury, the repair of a frontobasal CSF leak combined with that of maxillofacial fractures should be carried out as early as possible (Joss et al. 2001; Joseph et al. 2004).

7.2.4.1 Skullbase Fractures with Spontaneously Ceased CSF Leakage

If CSF leakage stops spontaneously, this may or may not indicate sufficient healing of the rupture. More so, the brain appears to seal off CSF flow temporarily. There

is evidence that spontaneous scar tissue only provides an insufficient barrier against recurrent leakage and infection (Probst 1971; Probst und Tomaschett 1990).

Spontaneously “healed” frontal rhinorrhea is reported to be followed by late meningitis and even brain abscess in 20–60% of cases (Paillas et al. 1967).

Older reports claim 24% morbidity and even 16% mortality with spontaneously healed CSF rhinorrhea (Lewin 1966, 1974). This is a strong argument in favor of surgical repair (Kästner et al. 1998).

7.2.4.2 Frontobasal Fracture with Suspected CSF Leakage

Frontobasal fractures without manifest CSF leakage may still be associated with a dural injury. Herniation of brain tissue, swelling of the nasal mucosa or blood clot may temporarily block CSF flow (Probst 1986; Fonseca and Walker 1991; Fonseca 2000). In significant frontobasal fractures with radiographic evidence of bone fragment dislocation, surgical repair should be considered even in the absence of manifest CSF rhinorrhea to avoid late complications, particularly meningitis (Watson 1967; Probst 1986; Schick et al. 1997).

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In the surgical management of complex maxillofacial fractures with frontobasal involvement, several important issues must be considered:

- Indication for surgery
- Surgical timing
- Surgical approach
- Surgical strategy
- Surgical technique

8.1 Indications for Surgery

8.1.1 Emergency Surgery (Probst 1971; Schwab 1995; Zink and Samii 1991; Schneider and Richter 1993)

From the sole neurosurgical perspective, surgery may be required as a life-saving procedure without delay (Schneider and Richter 1993).

Emergency surgical management will be indicated when there is:

- Evidence of raised intracranial pressure (ICP) due to epidural hematoma, subdural hematoma, intracerebral hematoma, cerebral contusion, and in rare cases due to intracranial air entrapment (Boenninghaus 1971; Loew et al. 1984)
- Life-threatening hemorrhage due to rupture of skull base vessels
- Open brain injury with exposed brain tissue

8.1.2 Semi-Selective Surgery for Frontobasal and Midface Fractures

Under certain circumstances surgery will be indicated on a semi-elective basis, allowing for time to treat more

urgent injuries or letting excessive swelling subside. Repair of a manifest frontobasal rhinorrhea falls under this category (Probst and Tomaschett 1990).

Indications are:

- Evidence of cerebrospinal fluid (CSF) leakage
- Significant pneumocranium with evidence for bone fragment dislocation
- Orbital complications with acute visual failure and/or double vision

Individual decision-making will to a certain degree inevitably depend on pragmatic evaluation of individual circumstances. Frontal skull defects, the degree of bone fragment dislocation, as well as the extent and timely reabsorption of intracranial air, may be considered to be surgical indications.

8.1.3 No Surgical Indication

Frontobasal fractures without dislocation and without evidence of CSF leakage.

8.2 Surgical Timing

Three aspects are critical for correct surgical timing (Samii et al. 1987):

- Evidence of raised ICP
- Frontobasal injury with CSF leak
- Presence of major craniofacial deformation

Frontobasal injuries frequently (68%) coincide with intracranial injuries (Probst and Tomaschett 1990):

- Intracranial hemorrhage and cerebral edema (41%)
- Injury to basal cerebral arteries (21.2%)
- Open brain injury (30.3%)

8.2.1 Evaluation

The timing and indication for surgical repair of craniofacial injuries depends upon the extent of the primary brain injury, as well as the anticipated development of secondary injury (cerebral edema, impairment of blood supply, inflammation, hyperexcitation, and seizures). Depending upon circumstances, the effects of secondary injury may be both mitigated and worsened as a result of early or late craniofacial repair.

- The assessment of individual circumstances and surgical timing always require the close interdisciplinary cooperation of the neurosurgeon and maxillofacial surgeon (Samii et al. 1995; Joss et al. 2001)

8.2.1.1 Neurosurgical Aspects

The primary factors to consider in surgical timing are the extent of direct cerebral injury, the degree of impaired consciousness (GCS), and the ICP (Piek and Jantzen 2000).

1. Intracranial injuries (cerebral contusion, cerebral edema, elevated ICP) determine the surgical timing in combined craniofacial fractures (Derdyn et al. 1990; Lausberg 1987; Metelmann et al. 1991; Hardt et al. 1992). Delaying surgery until after cerebral swelling has subsided was found to be preferable in terms of intra- and postoperative complications (Schroth et al. 1998).
2. Circumscribed intracranial lesions have to be addressed before the repair of frontobasal and maxillofacial injuries. Frontobasal and craniofacial reconstruction may be time-consuming and should thus be delayed until direct primary or secondary cerebral injury has begun to subside (Lausberg 1987; Piek and Jantzen 2000).
3. The cranial injuries themselves must be addressed according to the priorities listed in the previous chapter (Metelmann et al. 1991).

8.2.1.2 Maxillofacial Surgical Aspects

Surgical timing is primarily determined by the extent and severity of the traumatic damage. Soft tissue and osseous injuries, fracture dislocation and mainly the associated neurosurgical and ophthalmologic injuries indicate the necessity of an immediate intervention (Krafft et al. 1991; Metelmann et al. 1991; Zink and Samii 1991; Ewers et al. 1995).

1. The timing and sequence of surgical procedures, as well as the interdisciplinary coordination, must be individualized in each case depending on the severity of the injuries (Derdyn et al. 1990).
2. Maxillofacial reposition and stabilization procedures for complex craniofacial injuries should be performed simultaneously to the neurosurgical repair of frontal dural lacerations, to avoid disruption of dural grafts by maxillofacial interventions performed at a later stage (Joss et al. 2001). An unstable maxillofacial fracture may threaten any dural repair (Machtens 1987).
3. Early repair of maxillofacial fractures should be attempted to avoid bone healing in an incorrect position. Secondary surgery to correct midface fractures is more difficult and less satisfactory (Machtens 1987). For this reason, the maxillofacial surgeon tends to favor an early surgical approach.
4. The earlier surgical correction of craniofacial injuries is performed the smaller the surgical infection risk will be (Machtens 1987; Joss et al. 2001). According to international statistics (Buchanan et al. 2004; Joseph et al. 2004) “primary” surgical repair carries a statistical infection risk of between 4.6 and 7.1%, while late secondary surgery carries a risk of up to 17%. It appears that the concern about an increased infection risk due to combined intracranial, transoral, and transnasal approaches is unfounded (Götzfried et al. 1984).
5. Facial fractures with soft tissue injuries without skull base and/or dural injuries should be repaired in a single early surgical intervention whenever possible.

Specific time of surgery in cranial injuries subject to type of injury and state of consciousness (Lausberg 1987; Ewers 1995)

	Awake	Somnolent	Unconscious
Bone injury	Immediately	Temporize	Temporize
Intracranial air	Temporize	Temporize	Temporize
		Situational	Situational
Space-consuming hematoma	Immediately	Immediately	Immediately
Open cerebral injury	Immediately	Immediately	Immediately

8.2.2 Surgical Timing

The concept of early interdisciplinary treatment of maxillofacial and frontobasal injuries has been broadly accepted and applied in clinical practice (Gruss et al. 1985; Machtens 1987; Raveh and Vuillemin 1988; Gruss and Phillips 1989; Perrott 1991; Hardt et al. 1992; Evans et al. 1996; Lee et al. 1998; Joss et al. 2001).

8.2.2.1 Immediate Treatment (Phase 1) (Piek and Jantzen 2000)

Neurosurgical emergencies are craniofacial injuries combined with life-threatening injuries, particularly intracranial hemorrhage. In the majority of cases, a two-step approach will be appropriate, with the neurosurgical decompression being the immediate intervention and craniofacial surgery following stabilization and recovery (Zink and Samii 1991; Schneider and Richter 1993; Lehmann et al. 1998).

Maxillofacial surgical emergencies are significant uncontrolled hemorrhages from the skull base, significant soft tissue damage with tissue avulsion, open multifragmented maxillofacial and mandibular fractures, as well as optic nerve compression. The initial maxillofacial intervention may be confined to hemostasis and primary closure of soft tissues wounds (Fig. 8.1).

8.2.2.2 Primary Treatment (Phase 2) (Piek and Jantzen 2000)

Urgency, surgical timing and planning are determined by the individual extent of the injury and the extent of primary and secondary traumatic brain injury.

Open maxillofacial fractures should be operated within 6–8 h of injury (Metelmann et al. 1991):

- if there is only a moderate traumatic brain injury [Glasgow Coma Scale (GCS) > 8],
- if the patient remains stable upon GCS monitoring,
- if the control computed tomography (CT) scan shows no worsening

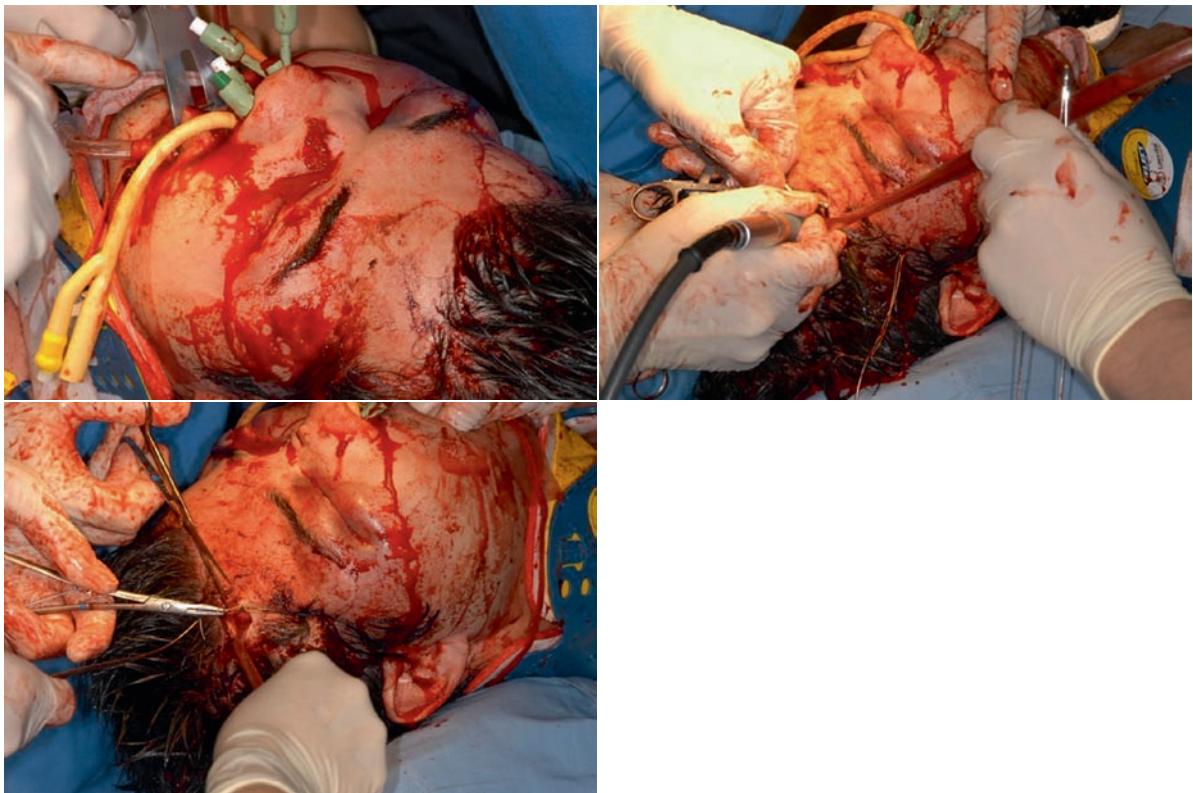


Fig. 8.1 Surgical intervention in the emergency room. Interskeletal stabilization of the midface in a patient with severe hemorrhage after complex midface fracture and frontobasal and cerebral injuries

This includes open fractures with and without frontobasal and brain injuries and open subcranial injuries.

Controlled ventilation, without significant hyperventilation, must be maintained during surgery, in order to avoid cerebral swelling. It may be useful to position the patient in a 30° head-up fashion. A 6-h time-frame should be maintained due to the significant swelling potential of facial soft tissues.

8.2.2.3 Elective Primary Treatment

Closed craniofacial and subcranial fractures with mild (GCS 14–15) to moderate (GCS 13–9) head injury may be treated within 12–24 h of injury.

These are:

- Craniofacial fractures without skull base involvement
- Craniofacial fractures with skull base involvement and severe head injury after stabilization of the neurological situation
- Subcranial midface fractures with/without frontobasal fractures

Craniofacial fractures and frontobasal fractures with significant fragment dislocation without significant brain injury should be treated within 12–24 h (Probst 1986).

Contraindications for elective primary treatment may be cardiorespiratory instability, coagulopathies, and other severe medical contraindications for a surgical intervention.

8.2.2.4 Delayed Primary Treatment

Patients with multiple severe injuries and patients with severe head injury (GCS < 8) and raised ICP should not be operated on before ICP remains consistently normal.

In patients with moderate head injury (GCS 13–9), neurological stabilization and normalization should be awaited before a decision for surgery is made (Dietz 1970a, b; Hausamen and Schmidseider 1975; Loew et al. 1984).

Too early surgical intervention may cause significant intraoperative brain swelling and subsequently may impair the surgical repair and adversely affect the neurological outcome.

- All craniofacial injuries with significant intracranial injury and raised ICP should only be treated after

normalization of ICP and cerebrovascular autoregulation (Metelmann et al. 1991; Zink and Samii 1991; Lee et al. 1998).

Delayed primary treatment for craniofacial and midface fractures with cerebral pathology should be postponed 5–10 days. Intracranial pressure, cerebral oxygenation, and cerebrovascular autoregulation must be recovered and intact. The presence of significant local brain contusion also warrants the delayed approach in order to avoid secondary brain swelling and hemorrhage (Probst and Tomaschett 1990).

An alternative to delaying surgery due to neurological instability may be surgery using a less invasive transfrontal-subcranial approach (Raveh and Vuillemin 1988).

8.2.2.5 Secondary Treatment

Complex injuries of both the craniofacial complex and the brain and dura may require a delay of surgical correction beyond 10 days, because neurological stabilization may take longer than this time-frame. Late treatment is faced with a technically much more difficult situation for repairing dislocated fractures.

Complex and multiple injuries pose a significant risk, so timing and surgical technique must be decided upon an individual basis (Schweiberer et al. 1987; Waydhas et al. 1997).

8.3 Surgical Approaches

The choice of the surgical approach to the craniofacial region and frontal skull base is based upon localization and extent of the skull base and midface injuries (Dieckmann and Hackmann 1977; Draf and Samii 1983; Samii et al. 1995; Joss et al. 2001).

Access should always be chosen in such a way as to enable an optimal approach and sufficient view of the skull base, upper midface region and participating orbital region for all disciplines involved.

Simultaneous care can be taken of midface fractures and injuries of the skull base with minimal additional access (Ernst et al. 2004).

The first priority is a complete exposure of all fractured regions without consideration of fragment size

(Dieckmann and Hackmann 1977; Hausamen and Schierle 2000).

The craniofacial and orbito-cranial regions including the skull base can be accessed through three principle approaches, depending on fracture localization and extent (Dieckmann and Hackmann 1977; Draf and Samii 1983; Samii and Brihaye 1983; Draf 1995; Lange et al. 1995; Samii et al. 1995; Steudel 1995; Ewers et al. 1995; Donald 1998; Eppey 2003)

- **Transfrontal approach**
Transfrontal-transcranial extradural approach
Transfrontal-transcranial intradural approach
Transfrontal-subcranial approach
- **Transfacial-frontoorbital approach**
- **Endonasal-endoscopic approach**

In the majority of cases, the *transfacial approach* is best-suited for unilateral localized revisions, whilst the *transfrontal approach* is mainly used for bilateral revisions in skull base fractures and in concomitant

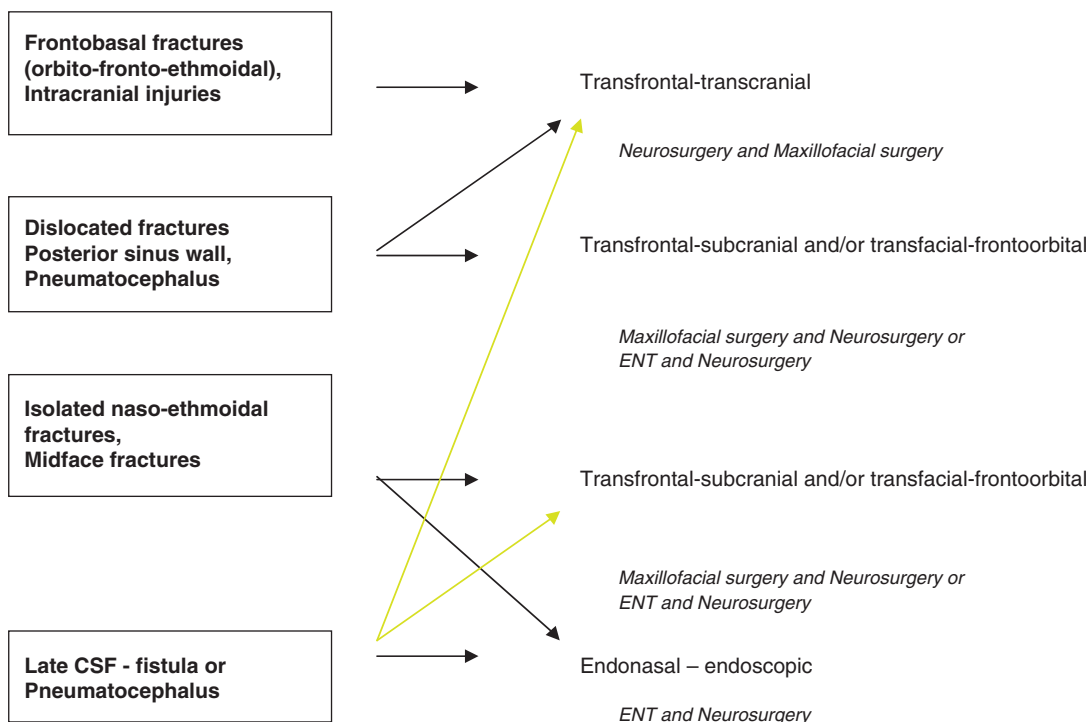
fractures of the fronto-facial compartment, as well as fractures of the upper midface with dislocated base injuries (Brisett and Hilger 2005) (Fig. 8.2).

8.3.1 Strategy for Interdisciplinary Approach (Decision Criteria)

The criteria in order to determine the interdisciplinary surgical approach in combined midface- and skull base fractures result, on the one hand, from the *injury pattern* (Samii and Brihaye 1983; Samii 1987; Zink and Samii 1991; Waydhas et al. 1997) and, on the other hand, from the *localization* and extent of the injury (Müller et al. 1996; Lehmann et al. 1998; Brisett and Hilger 2005).

Decision criteria for the interdisciplinary treatment of combined midface-skull base fractures, depending on localization and extent of the fractures (mod. a. Lehmann et al. 1998):

Decision criteria for the interdisciplinary treatment of combined midface-skull base fractures, depending on localization and extent of the fractures
(mod. a. LEHMANN et al. 1998)



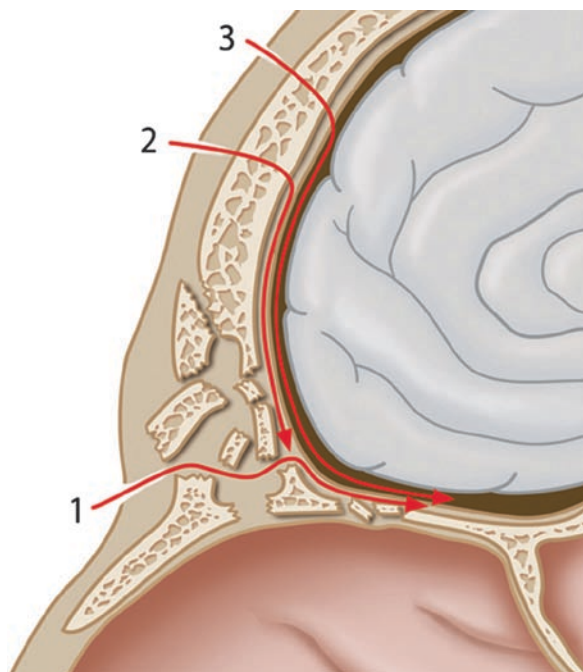


Fig. 8.2 Different approaches to the frontofacial and frontobasal region (mod. a. Ewers et al. 1995). 1 Transfacial, 2 transfrontal-transcranial-extradural, 3 transfrontal-transcranial-intradural

8.3.1.1 Approach Strategy: Transfrontal-Transcranial

Complex craniofacial injuries with extensive orbito-fronto-ethmoidal fractures should be treated from the *transfrontal* aspect (Vuillemin et al. 1988; Giuliani et al. 1997; Steudel 1995). The *extradural* approach to the skull base involves transection of the olfactory fibers if the cribriform plate has to be exposed bilaterally. Using the extradural approach, it is possible to preserve the olfactory fibers when exposing the posterior wall of the frontal sinus and anterolateral skull base (Samii 1989; Samii et al. 1989; Lehmann et al. 1998).

In severe comminuted fractures with multiple and extensive dural disruptions, the *intradural* approach is used, particularly as dural disruption traverses the region of the cribriform plate, the roof of the ethmoidal cells and crista galli. This is particularly the case in base injuries – whichever localization – with increasing intracranial dislocation or swelling of the brain with a threatening tentorial incarceration (Samii 1989).

Depending on the fracture dimension, *deep fronto-basal fractures* resulting from a force applied to the mid-face (type III-Escher) are treated extradurally through the subcranial or the transcranial approach. This type of fracture, especially, necessitates a functionally stabile reconstruction of the midface as a prerequisite for neurosurgery (Schwab 1995; Ernst et al. 2004).

8.3.1.2 Approach Strategy: Transfacial-Frontoorbital or Transfrontal-Subcranial

As an alternative to the transfrontal approach, the *transfacial approach* lends itself to treating localized injuries in the ethmoido-frontal sinus region. The *transfrontal-subcranial approach* in subcranial mid-face fractures with dislocation of the posterior frontal sinus wall or naso-ethmoidal fractures should only be applied if the nasal base is not traversed and if there are no severe cerebral injuries (Vuillemin et al. 1988; Lehmann et al. 1998).

The *transfacial or transfrontal subcranial* extradural approach is used to expose the fractures and dural injuries. Subsequently, treatment of the upper paranasal sinus and midface fractures follows (Boenninghaus 1974; Schwab 1995).

The *transfacial frontoorbital* approach can be applied in localized mid-frontobasal fractures resulting from circumscribed forces against the forehead/nasal base region (type II-Escher). Fracture debridement and cleansing of the upper paranasal sinuses are carried out, with skull base exploration to ensure an uninjured dura. Subsequently the midface fractures are treated (Boenninghaus 1974; Schwab 1995).

Latero-orbital fractures of the frontal bone (type IV-Escher) are treated either by a *transfacial frontoorbital approach* or, in the case of considerable dislocation of orbital roof parts (the zygomatico-temporal region) by a *transfrontal-extracranial* approach (Schwab 1995).

8.3.1.3 Approach Strategy: Endonasal-Endoscopic

The endonasal-endoscopic approach is reserved for isolated ethmoidal and sphenoidal fractures with dural injuries and recurrent liquorrhea (Stoll 1993). This technique is widely used by ENT-surgeons.

8.4 Transfrontal-Transcranial Approach

In craniofacial injuries with a large degree of intracranial fragment dislocation and/or multiple dural injuries, it is generally advised to use the transfrontal-transcranial approach (Gruss 1982; Westmore and Whitam 1982; Myers and Sataloff 1984; Strohecker 1984; Loew et al. 1984; Manson et al. 1985; Probst 1986; Probst and Tomaschett 1990; Hardt et al. 1992; Hausamen and Schmelzeisen 1996; Joss et al. 2001).

The transfrontal-transcranial approach:

- Allows simultaneous treatment of accompanying intracranial and basal injuries. In particular frontobasal fractures, and defects can be repaired with variable sized pericranial flaps and/or bone grafts (Schilli and Joos et al. 1991; Hardt et al. 1992; Joss et al. 2001).
- Facilitates the paramount primary reconstruction of the fronto-facial compartment and the zygomatic complex — the key structure for the subsequent alignment and osteosynthesis of the midface (Sailer and Gratz 1991; Hardt et al. 1992; Prein 1998).
- Ensures stable osteosynthesis of the subcranial structures and an optimal protection of the repaired dural- and frontobasal injuries (Joos and Gilsbach 1992; Joos et al. 1989; Schilli and Joos 1991; Hardt et al. 1992; Weingart et al. 1996; Kessler and Hardt 1998a, b; Donald 1998; Joss et al. 2001).
- Simultaneously, autogenous bone grafts can be harvested from the calvarium using the split-skull technique and used to cover basal or frontal defects (Manson et al. 1985).
- The wide bifrontal incision lies in an aesthetically inconspicuous region.

8.4.1 Indications

The indications for a transcranial approach largely depend on radiological findings. The cranial CT (CCT) must either display a basal fragment dislocation or indirect evidence of a dural injury (Whitaker et al. 1998).

- (a) In *complex craniofacial fractures with simultaneous frontobasal fractures*, the transfrontal approach offers the possibility of concurrently treating the fractures either via the transfrontal-extradural or the subcranial-subdural access (Raveh and Vuillemin

1988; Krafft et al. 1991; Hardt et al. 1993; Prein 1998). Indication for the intracranial method is derived from the fact that *multiple dural fistulas* can be demonstrated in 44% of skull base fractures; 2% were localized bilaterally, so making an exposure of the total frontal base essential (Probst and Tomaschett 1990).

- (b) *Subcranial midface fractures* (Le Fort II and III) *without skull base fractures* often necessitate the transfrontal approach in addition to the conventional facial approach, particularly for the correct three-dimensional reposition and reconstruction of the naso-ethmoidal complex, as well as the zygomatic bones and zygomatic arches (Prein 1998).
- (c) In the case of *unilateral complex lateral midface fractures*, the transfrontal approach may become necessary for correct reconstruction of the midface (Weber and Michel 1989; Prein 1998).
- (d) Difficulties in treating the *peri- and interorbital region in central midface fractures* can be avoided by using the transfrontal-subcranial procedure (Raveh et al. 1984, 1992, 1993, 1998).

8.4.2 Transfrontal-Transcranial Approach: Surgical Technique

8.4.2.1 Coronal Approach

The transfrontal approach is carried out with a coronal incision according to Unterberger (1959) (see also Tessier 1971; Prein 1998; Eppley 2003). In contrast to transfacial approaches, the coronal approach renders perfect exposition of the cranio-orbital region, the frontal base, the periorbital- and zygomatic region (Weber and Michel 1989; Krafft et al. 1991; Rohrich and Hollier 1992; Hardt et al. 1993; Prein 1998).

Advantages of the coronal approach (Shepherd et al. 1985; Schilli and Joos 1991; Hardt et al. 1993; Haerle et al. 1999) are:

- Protection of the epicranial vascularization
- Formation of a variably formed pedicled pericranial flap
- Broad exposure of frontal, zygomatico-orbital, and periorbital structures
- Exposure of the parietal region to gain autogenous calvarian bone grafts

The transfrontal approach classically results from a *coronal incision*. The skin is cut from ear to ear above the

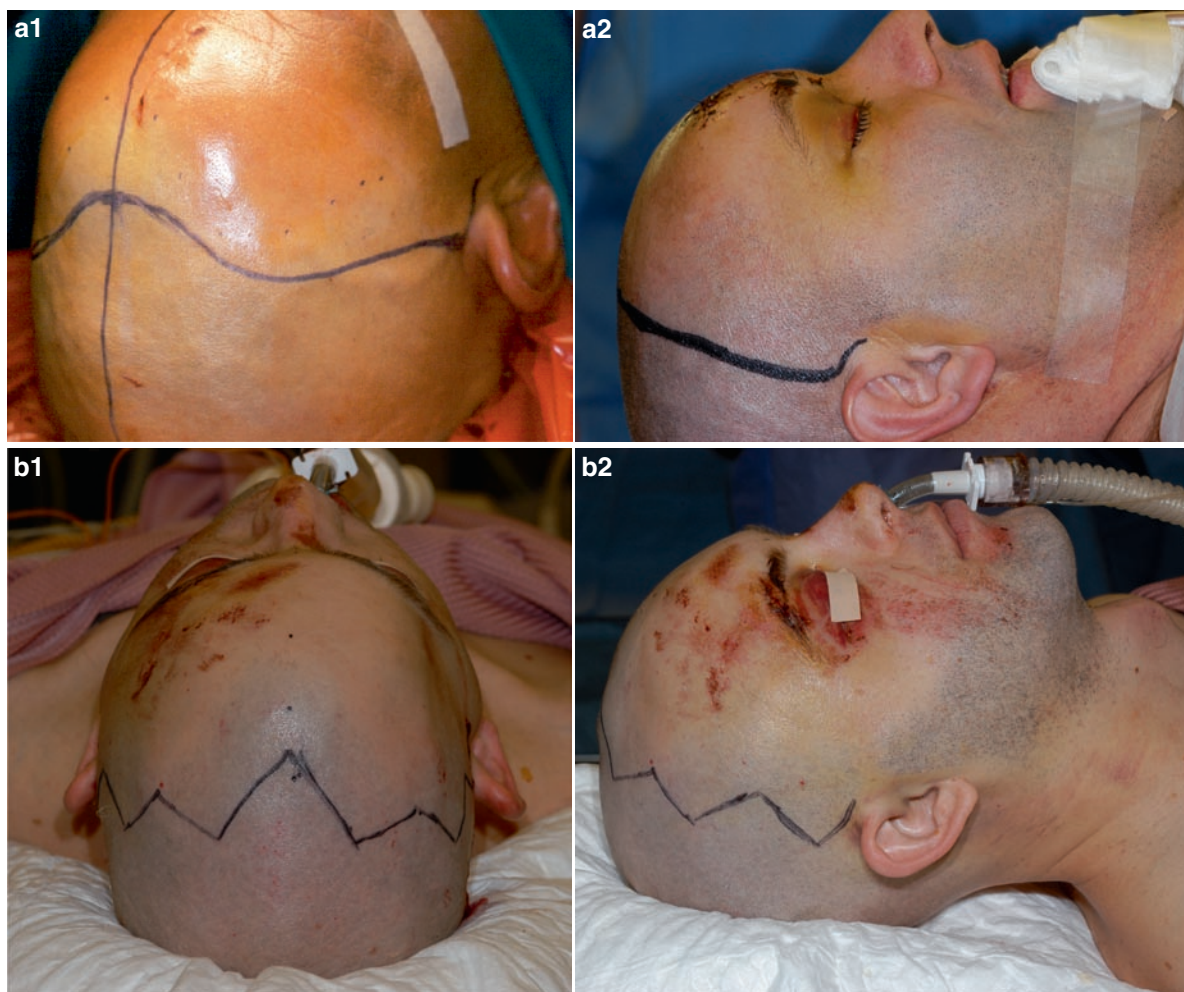


Fig. 8.3 Coronal approach (types of skin incision). (a) Classic wave-line incision. (b) Angled/zig-zag incision to make the scar less noticeable

temporal fascia, bilaterally as far down as the zygomatic arch. The incision runs approximately 3–4 cm behind the frontal hairline. Using a *preauricular extension* of the coronal incision the zygomatic arches and the zygomatic complex can be clearly exposed (Shepherd et al. 1985; Fonseca and Walker 1991; Sailer and Gratz 1991; Kellman and Marentette 1995). The preauricular incision runs along the border of the tragus or behind the tragus (Eppley 2003).

As a variation, the semilunar incision can be replaced by an *angled incision* (W-incision/zig-zag incision) (Hammer and Prein 1998; Eppley 2003). The W-incision improves the readaptation of the wound margins and the scar is much more inconspicuous compared with the straight incision (Eppley 2003) (Fig. 8.3).

Depending on the pattern of injury, further incisions or variations may be necessary. In case of a *frontal soft tissue*

wound, this must previously be closed. Subsequently the coronal incision can be made. If an extensive comminuted zone with a large skin defect exists, the coronal incision has to be appropriately modified (Probst 1971).

- Coronal approach: Technical points

In carrying out the coronal approach, consideration must be taken of the soft tissues, fascial layers and the neural structures, as the frontal branch of the facial nerve runs within the temporal fascia (Weerda 1995; Haerle et al. 1999) (Fig. 8.4).

- Preparation of skin/galea and pericranial flaps

The skin incision is performed from ear to ear above the temporal fascia, bilaterally 2–4 cm behind the hairline. The incision divides the skin, subcutis, and galea, though not the pericranium and the temporal fascia.

Fig. 8.4 Schematic diagram of the different layers of the skull (mod. a. Weerda 1995).
 1 Cutis, 2 subcutis, 3 galea aponeurotica, 4 subgaleatic layer, 5 pericranium, 6 bone, 7 dura mater

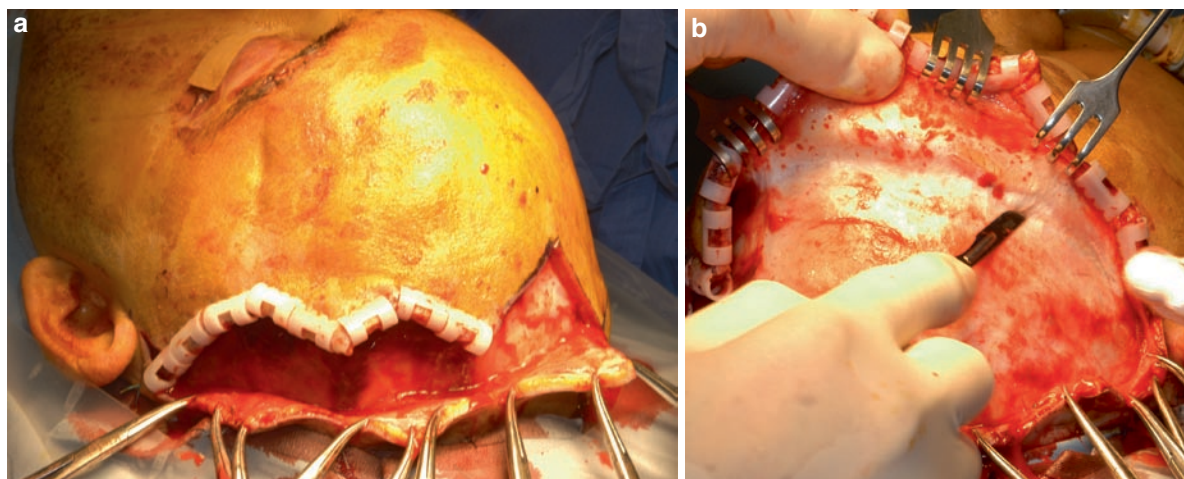
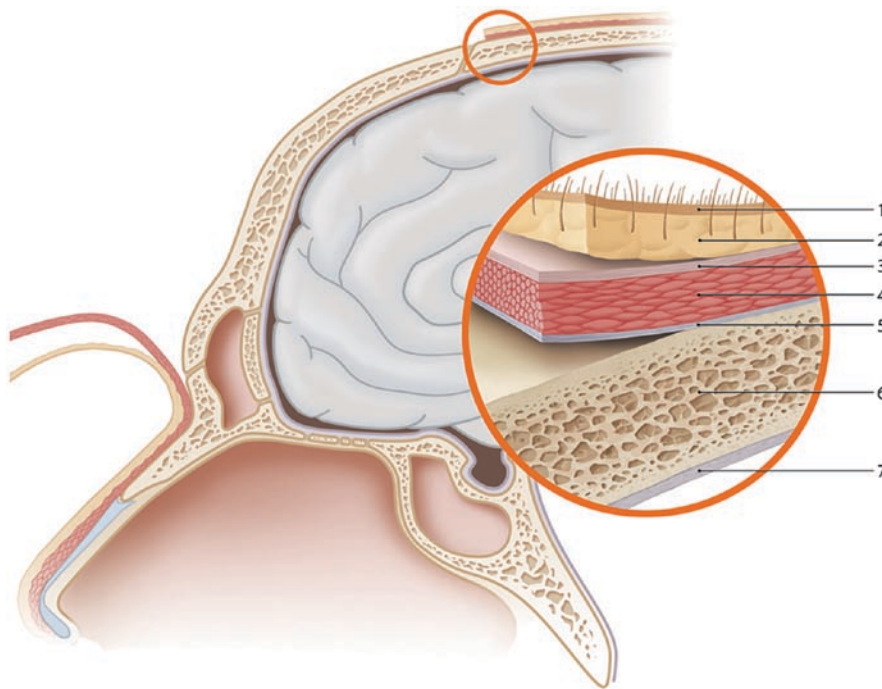


Fig. 8.5 Coronal approach: dissection of the skin-galea-flap. (a) Incision and hemostasis with Dandy clamps and Rainey clips. (b) Sharp dissection in the subgaleal plane. The subgaleal dissection stops about 2 cm above the supraorbital rim

Hemostasis is carried out along the incision by Raney clips or with Dandy clamps. We avoid cutting with the electric knife as well as extensive bipolar coagulation along the incision borders. After transecting the subcutis, one can either precede epiperiosteally or the periosteum is transected and one then proceeds subperiosteally. In the region of the temporal muscle, dissection is carried out directly on the deep fascia of the temporalis muscle (DTF) to avoid injury to the frontal branch of the facial nerve (Fig. 8.5).

The skin-galea flap is removed *epiperiostally* (subgaleatic gliding layer) from the underlying pericranium up to approximately two finger widths (~3 cm) above the supraorbital rim. When the skin-galea flap has been dissected, it can be turned over the face and fixed. To dissect a *pericranial flap* the pericranium has to be incised and is elevated subperiosteally. The pericranial flap is well perfused due to its pedicle in the supraorbital region. The dissection goes as far as to the supraorbital margin. The periorbital, nasal, and glabella region

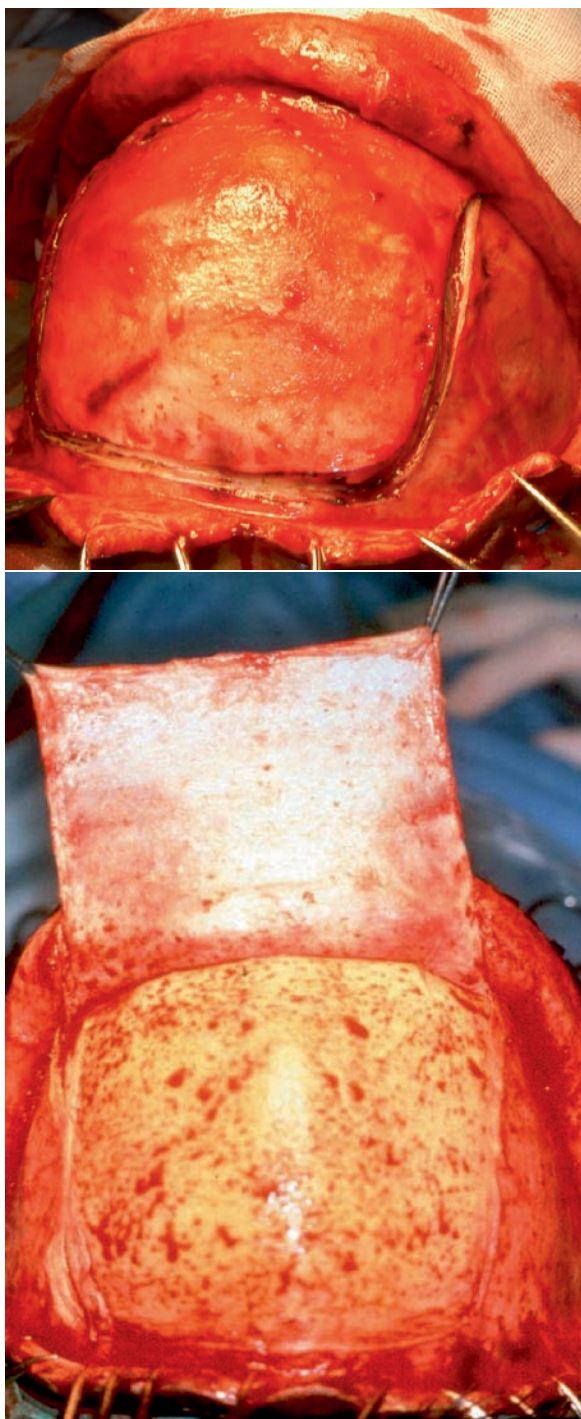


Fig. 8.6 Coronal approach: incision and dissection of the pericranium flap. After incision and mobilization of the pericranium flap about 2 cm above the orbital rim, the supraorbital rim is exposed subperiostally

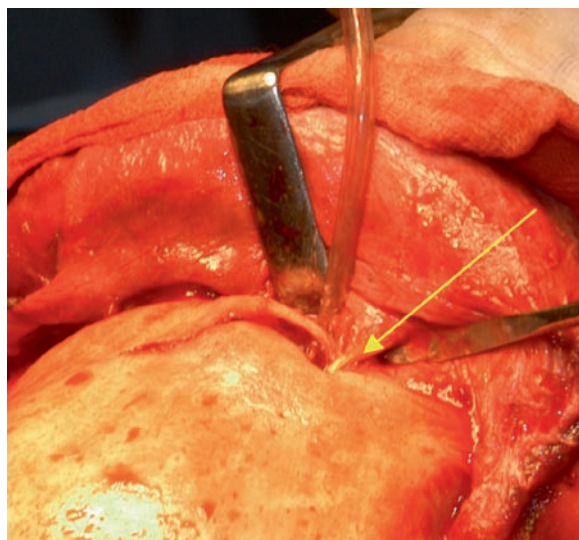


Fig. 8.7 Coronal approach: subperiosteal exposure of the supraorbital rim. Identification, dissection, and release of the supraorbital neurovascular bundle by removal of the bone inferior to the supraorbital foramen (arrow)

is exposed, including the neurovascular supraorbital bundle (Hardt et al. 1993).

In order to protect the frontal and supratrochlear neurovascular bundles, they should be released from their foramina and included in the skin flap (Ewers et al. 1995). The bipedicle flap can easily be removed from the bone as far as the root of the nose or even the lateral orbital wall up to the lower margin of the orbit (Figs. 8.6 and 8.7).

The degree of exposition of the orbital region or the entire nasal pyramid that can be achieved is primarily dependant on the type of lateral preauricular incision and the preparation of the flap above the temporal muscle.

There may be an occasional anatomical difficulty with the exposure of the root of the nose. A prominent frontal bone and a deep naso-frontal suture may narrow the operative field in the intraorbital region when applying the coronal incision alone. In such cases, we complete the incision with a partial fronto-orbital or median nasal access (Hardt et al 1993).

- Exposure of the lateral orbital wall

Injuries to the temporal branch of the facial nerve should be avoided during the exposure. The frontal branch is situated in the superficial movable layer of

the superficial temporal fascia (STF) lateral and above to the outer fascia of the temporalis muscle. After subgaleatic dissection and exact epifascial preparation in the temporal muscle region between the STF and the outer layers of the deep temporal fascia, the incision of the pericranial flap is performed. A curved incision following the origin of the temporal muscle is made, beginning approximately 2 cm dorsal of the lateral orbital margin. Here, the subfascial space under the deep temporal fascia is opened and the temporal fat pad is identified. In this plane the preparation towards the lateral orbital wall is performed, so protecting the frontal branch of the facial nerve (Imhof 2000; Eppley 2003).

- Exposure of the glabello-nasal compartment

The periosteum and the periorbit are precisely dissected subperiostally in the region of the orbital margin, as here tearing may easily occur and bulbar fatty tissue may protrude. Exposure of the naso-frontal region and supraorbital nerve is facilitated by a median vertical periosteal incision. With progression, the entire midface can be exposed down to the lachrymal sac and cartilaginous nose (Hardt et al. 1992). This extensive dissection can be facilitated by continuing the coronal incision bilaterally down to below the tragus.

Exposure of the glabello-nasal compartment (Kellman and Marentette 1995)

- Release the supraorbital neurovascular bundle
- Median periosteal incision to expose bony nasal structures
- Exposure of the naso-orbital structures

- Exposure of the orbital walls

After reaching the supraorbital margin, the supraorbital neurovascular bundle is bilaterally released from its osseous foramen. This is followed by circular subperiosteal dissection of the orbit, providing a good view of the fracture and an insight of its extent in the orbital wall and especially the medial naso-orbital region.

- Exposure of the zygomatic complex

In case additional access to the zygomatic bone and zygomatic arch complex is necessary, the following is undertaken. After an arcuate or diagonal incision of about 45–30° in the deep temporal fascia (DTF) directed to the tragus, one proceeds interfascially between both layers of the deep temporal fascia, exposing the temporal fat pad, towards the zygomatic structures (Kellman and Marentette 1995; Politi et al. 2004). The frontal

branch of the facial nerve runs superficial to the STF. Using this subfascial approach, the zygomatic complex can be exposed without endangering the frontal branch of the facial nerve (Haerle et al. 1999).

Technique in exposing the zygomatic complex (Kellman and Marentette 1995)

- Incision (30–45°) in the STF and exposure of the outer layer of the deep temporal fascia
- Arcuate or diagonal transection through the outer layers of the deep temporal fascia
- Subfascial preparation between fascia and fat pad on the zygomatic arch and periosteal incision
- Subfascial/subperiosteal exposure of the zygomatic body from dorsal to ventral

If necessary for the subsequent craniotomy, the temporal muscle will be detached from the temporal bone with a broad periosteal elevator (Figs. 8.8–8.11).

- Complications associated with the coronal approach

One has to differentiate between surgically-induced and trauma-induced complications (Shepherd et al. 1985). Hardt et al. (1992, 1993) experienced the following complications induced by the surgical approach:

- Sensory dysfunction

Apart from direct pre- and intraoperative injuries, an extensive strain to the neurovascular bundle can be responsible for a temporary sensory loss, especially if it is not released from its foramen intraoperatively.

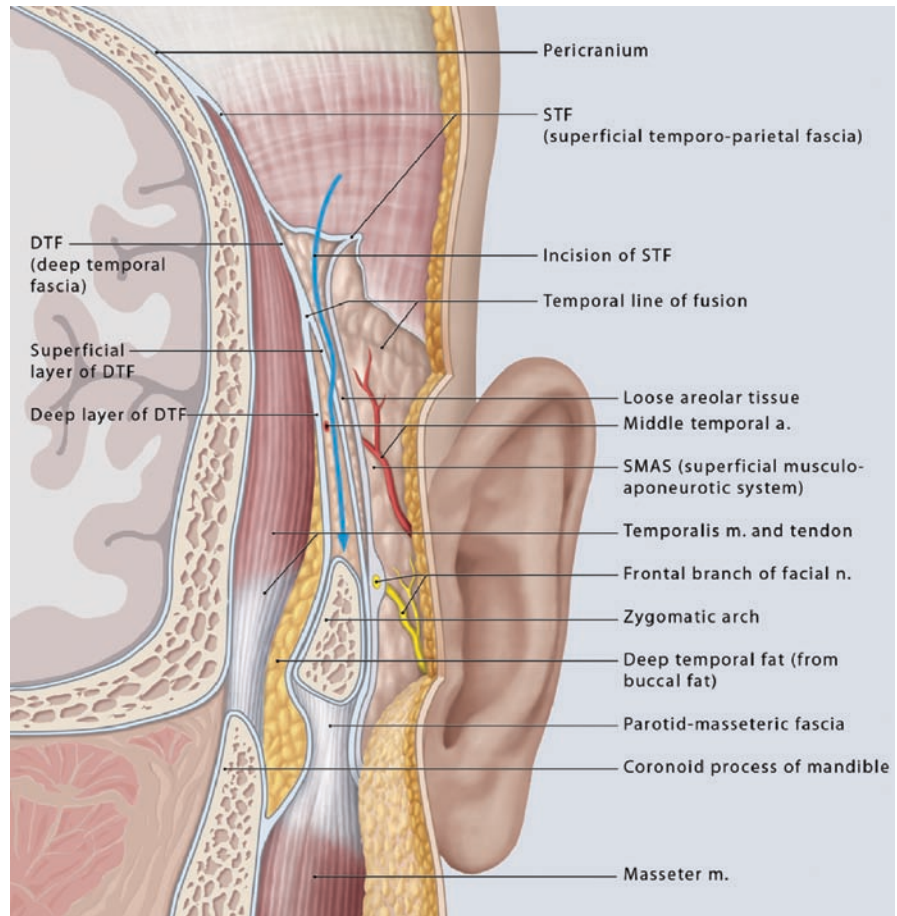
- Dysfunction of the supraorbital nerve (V1) in 7% (temporary paresthesia)
- Definite sensory loss due to injury (V1) in 2% (permanent anesthesia)
- Motor dysfunction

Temporary weakness of the frontal branch of the facial nerve was found unilaterally in 14% and bilaterally in 4%. Permanent dysfunction remained in 2% of the unilateral cases. Whilst the temporary dysfunction can be accounted for by distension, the permanent deficiency is without doubt due to intraoperative neural injury.

- Subgaleatic hematomas

Extensive subgaleatic hematomas were evident in 4% of the cases following intraoperative graft removal from the temporal muscle. This can be avoided by meticulous hemostasis at the donor site and compressive taping of the wound after wound closure.

Fig. 8.8 Coronal approach: planes of dissection to the zygomatic complex (mod. a. Kellman and Marentette 1995). The subgaleal dissection over the temporal muscle continues epifascially on the surface of the STF and stops superiorly to the temporal line of fusion of the superficial and deep layer of the DTF in order to preserve the frontal branch of the facial nerve. The superficial temporo-parietal fascia is incised, and dissection continues under the SFT fascia. Below the temporal line of fusion, the dissection has to be done directly under the superficial layer of the DTF



8.4.2.2 Osteoplastic Craniotomy

A classical approach to the frontal skull base can be gained by a uni- or bifrontal craniotomy (Unterberger 1959; Schmidek and Sweet 1988; Imhof 2000). This approach enables the surveillance of the entire anterior skull base, including the orbital roofs, cribriform plate, ethmoid, and frontal sinuses. The drill holes are positioned individually according to the line of fracture. The craniotomy is either bifrontal-symmetrical or in favor of the more affected side.

The craniotomy holes should be connected in such a way that the parasagittal holes are connected last, so that in case of an iatrogenic or existing injury to the superior sagittal sinus, access to the sinus is assured within a short time. It is necessary to carefully and subtly release the dura with special dissectors before lifting the depressed fragments, to avoid tears to the dura and the venous drainage system (Figs. 8.12–8.14).

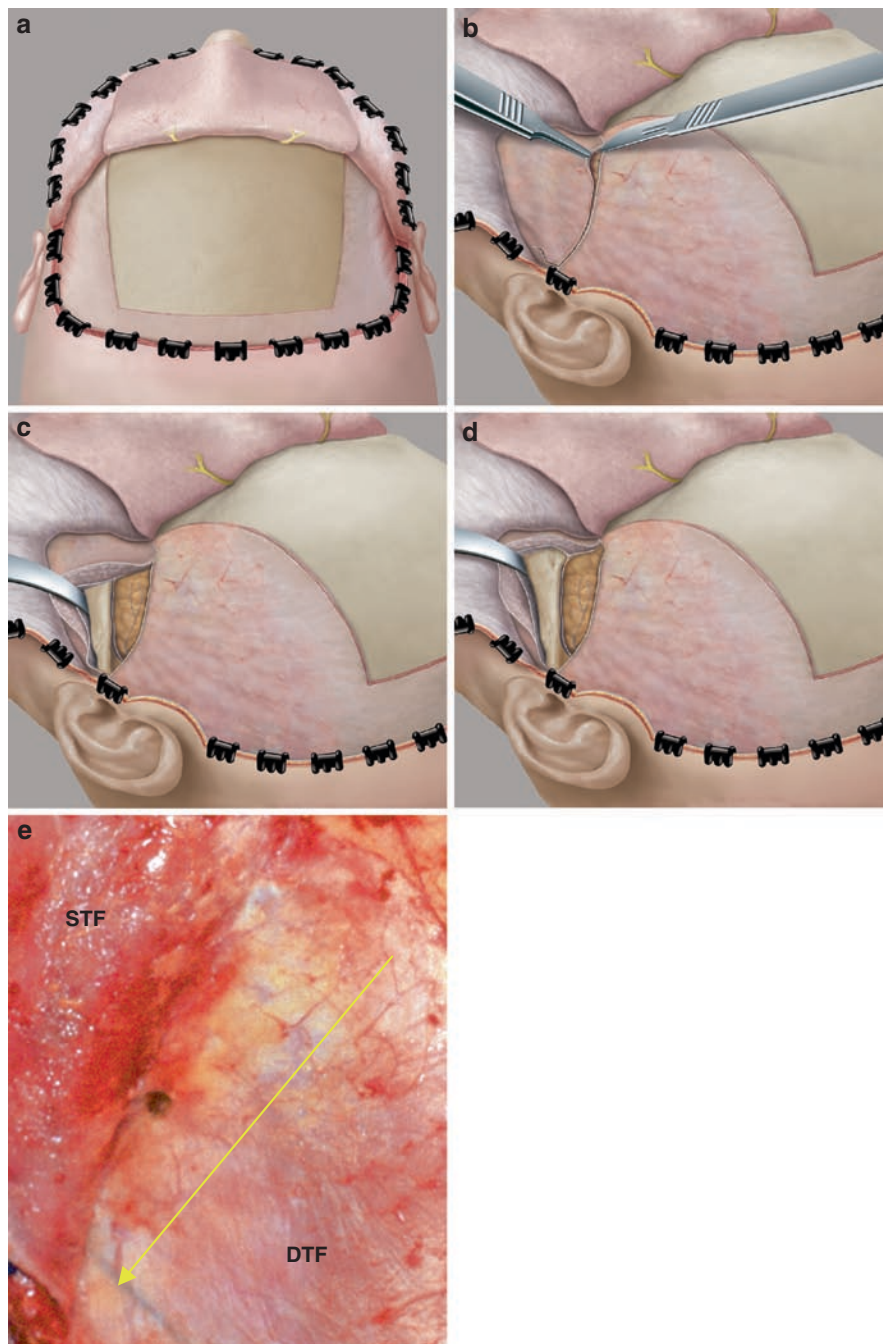
Hemorrhage originating from the venous sinus system is primarily best controlled digitally. Subsequently the defect is sealed by compression through a muscle patch, which is secondarily reinforced by bridging sutures and application of fibrin glue.

Using the intradural access, an additional bore hole to the temporal bone and to the superior temporal line not only enables the inspection and revision of the inferior surface of the frontal lobe from anterior, lateral, and from the wing of the sphenoid bone but also that of the optic nerve, internal carotid artery, basal cisterns and the orbital fissures as well as the temporal pole and the inferior surface of the anterior third of the temporal lobe (Imhof 2000).

- Frame-like craniotomy

After detaching the pericranium, four bore holes are drilled around the fracture site. The dura between the burr holes is separated from the internal table with a

Fig. 8.9 Coronal approach: exposure of the zygomatic complex (mod. a. Kellman and Marentette 1995). (a) The superficial layer of the DTF is incised at the root of the zygomatic arch. The incision continues at an angle of 45° until it joins the cut edge of the pericranium flap. To facilitate exposure of the zygomatic arch, a preauricular extension of the incision can be performed. (b) Incision of the superficial layer of the deep temporal fascia exposes the superficial temporal fat pad. The dissection is then carried out inferiorly between the fascia and superficial to the fat pad until the zygomatic arch and the posterior border of the zygoma are reached. This plane of dissection provides a safe route to the zygomatic arch because the temporal branch of the facial nerve is retracted laterally with the superficial layer of the deep temporal fascia. (c) The zygomatic arch is initially approached at its root, where the periosteum is incised along the superior border. The incision continues anteriorly along the posterior border of the zygoma and reaches the cut edge of the pericranium flap. (d) A subperiosteal dissection is then performed, which exposes the lateral surface of the zygomatic arch, the body of the zygoma and the lateral orbital rim (e) Intraoperative situation (arrow: diagonal line of incision in the superficial layer of the deep temporal fascia (DTF))



narrow, blunt dissector and the fractured calvarial bone plate is sawed out with a 'Gigli' saw, mobilized, and removed. Bone dust is collected (Imhof 2000).

- Initial fragment stabilization

In polyfragmentation without dislocation of the frontal calvarium, stabilization of the calvarian pieces with miniplates should antecede a craniotomy. If bone

transplants are necessary to cover defects, lamina interna bone grafts can be gained from the craniotomized and split bone cap.

- Mobilization from within a bore hole

If large pieces of the calvarium are impressed and the fragments difficult to mobilize, then a hole is drilled on the outer area of the fracture site and widened

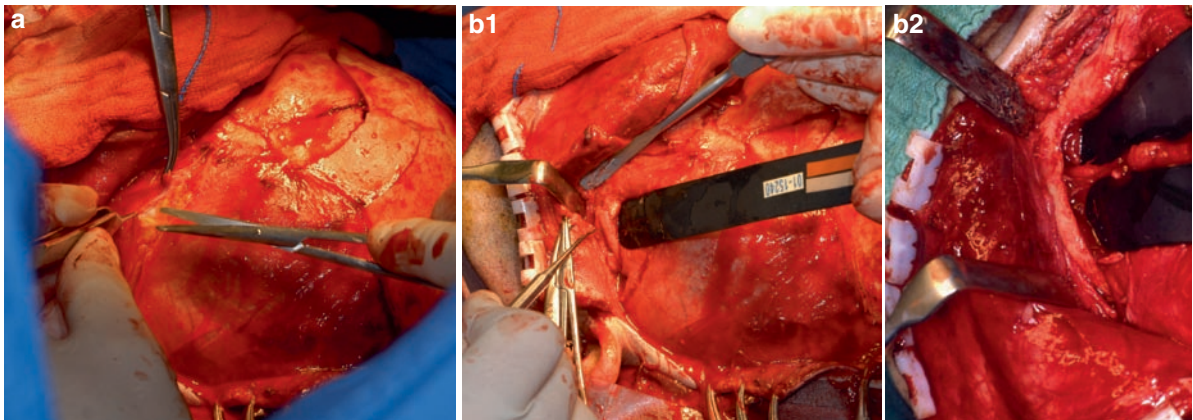


Fig. 8.10 Coronal approach: exposure of the zygomatic complex. (a) Incision of the superficial layer of the DTF at a 45° angle and subfascial dissection superficial to the temporal fat pad.

(b) After incision of the periosteum the zygomatic arch is exposed in a subperiosteal plane

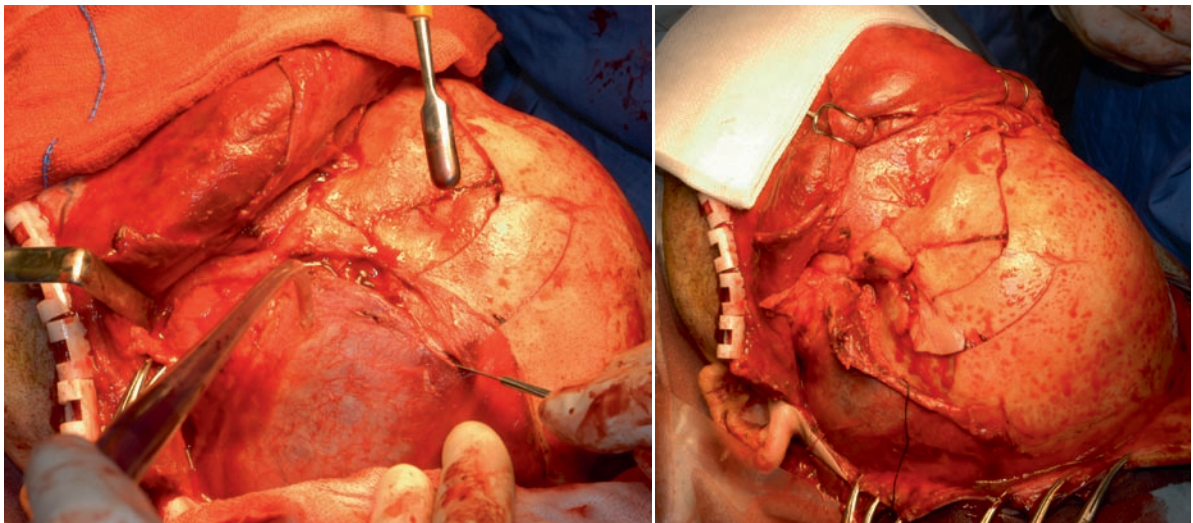


Fig. 8.11 Coronal approach: exposure of the pterional region. Incision and elevation of the temporal muscle to expose the pterional region

osteoclastically (with a rongeur) up to the edge of the impressed region. Working from the drill hole, one of the nearest fragments can be separated from the dura, mobilized, and removed. This process is repeated for the remaining fragments.

All fragments are carefully preserved, reassembled exactly outside the operative field and stabilized with miniplates to be reintegrated later as a calvarian transplant.

- Extension osteotomies

For an improved frontobasal exposure, additional fronto-facial segments can be osteotomized to facilitate a direct

base-parallel approach (Hardt et al. 1992). These fronto-facial osteotomies can vary due to location and extent of the fracture site.

Frontal extension - osteotomy

- Applying vertical and horizontal frontofacial osteotomies with a micro-saw
- Removal of the cranio-frontal or entire frontofacial segment

By selectively osteotomizing the cranio-orbital or cranio-frontal bone compartments (supra-orbital

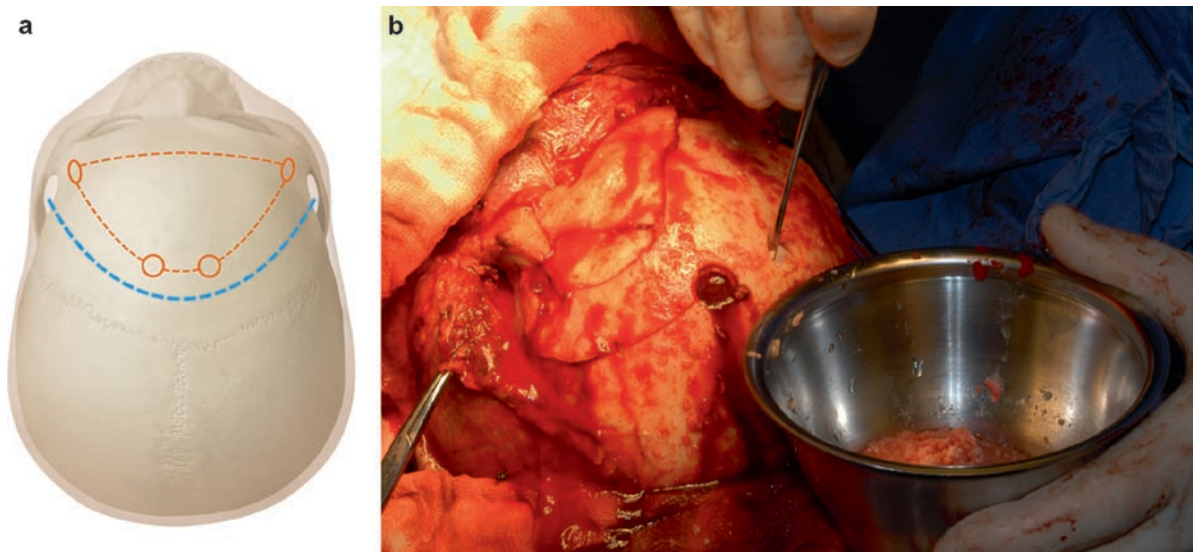


Fig. 8.12 Transfrontal craniotomy. (a) Design and position of the burr holes in a typical bifrontal craniotomy (blue: incision). (b) Bifrontal craniotomy. The paramedian burr holes are placed lateral to the superior sagittal sinus. Bone dust is collected

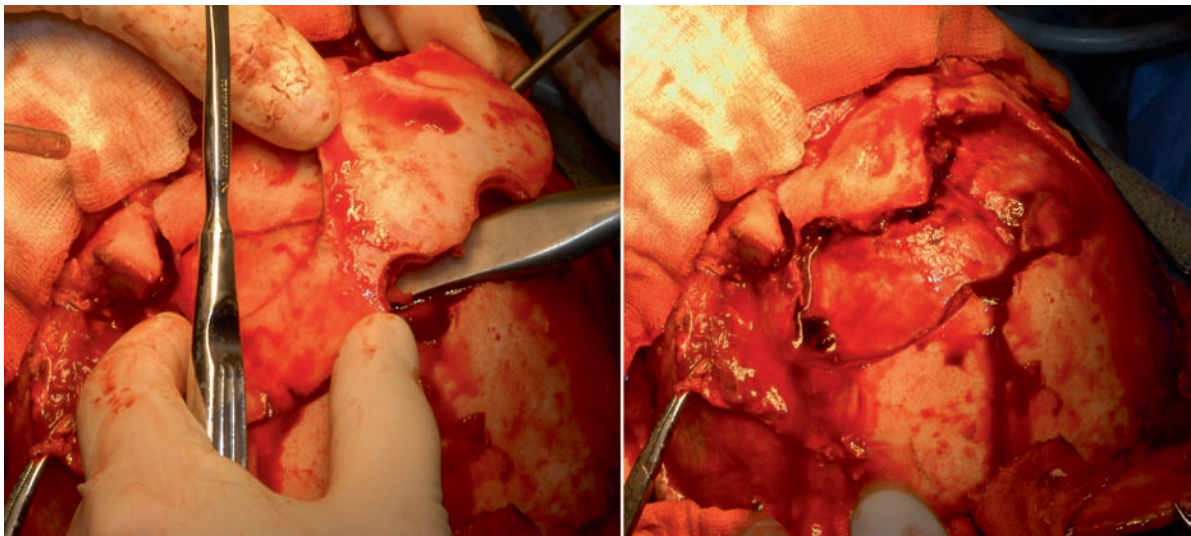


Fig. 8.13 Elevation of the frontal bone flap, dissection of the dura and exposition of the fronto-temporal area

bandeau) and after carefully extradurally raising the frontal cerebral lobe, one obtains a subfrontal tangential view of the whole frontal skull base – if possible, selectively protecting the olfactory fibers.

This osteotomy provides an improved exposure of the dural injuries without undue retraction of the frontal lobe (Hardt et al. 1992; Kessler and Hardt 1998a, b; Kuttenger and Hardt 2001). With regard to the level of

access, this procedure is approximately equivalent to that of the subcranial-subdural approach (Figs. 8.15–8.17).

8.4.2.3 Skull Base Exposition

One differentiates between an *extradural* and an *intradural* exposition, depending on whether or not the dura

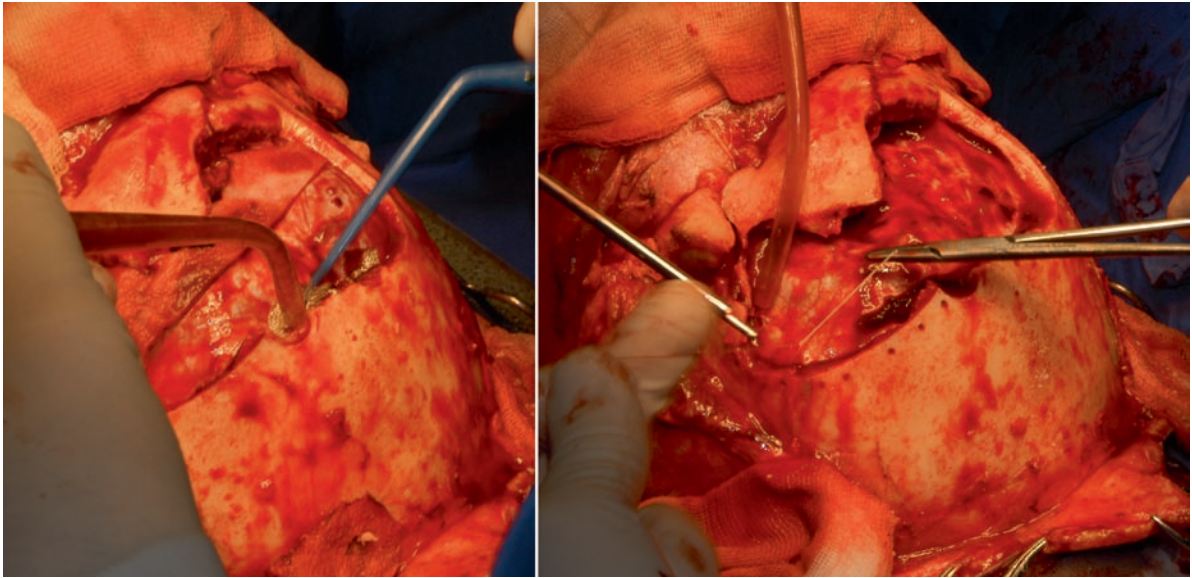


Fig. 8.14 Hemostasis is performed using hemostytic gauze, bipolar cautery and application of tack-up sutures

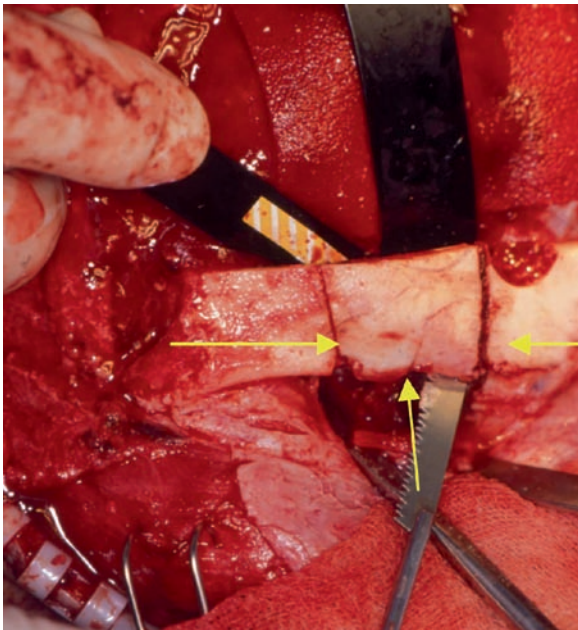


Fig. 8.15 Osteotomy of the nasofrontal segment to facilitate horizontal exposure of the medial frontobasal region without traumatic brain retraction. The horizontal and vertical osteotomies are performed using a microsaw (*arrow*)

is raised from the basal bone. The decision as to whether to operate intra- or extradurally, or even through both approaches if necessary, must be made by the neurosurgeon intraoperatively. Crucial criteria are extent and

localization of the dural injuries. If there is no intradural pathology or trauma in need of revision, then fractures in the region of the skull base, frontal sinus or the orbital roofs are revised via a transfrontal-extradural approach.

- Extradural exposition of the skull base

Extradural exploration is particularly applicable in all basal injuries in the vicinity of the frontal sinus, anterior, and mid-ethmoid and orbital roof with no cerebral injury. Debridement of the anterior and mid-ethmoid region and consequently the frontal sinus can be carried out from the epidural space (Giuliani et al. 1997).

Technical Aspects

After removing the skull bone and the fronto-facial fragments, one can expose the floor of the anterior cranial fossa. Using light-enforced dura dissectors and bipolar Malis forceps, the dura is carefully peeled off from the floor of the anterior cranial fossa and the orbital roofs as far as the edge of the greater wing of the sphenoid bone.

In doing so the dura is also relieved antero-medially towards the crista galli. Should it be necessary to expose the cribriform plate bilaterally, then the falx must also be removed, possibly in combination with resection of the crista.

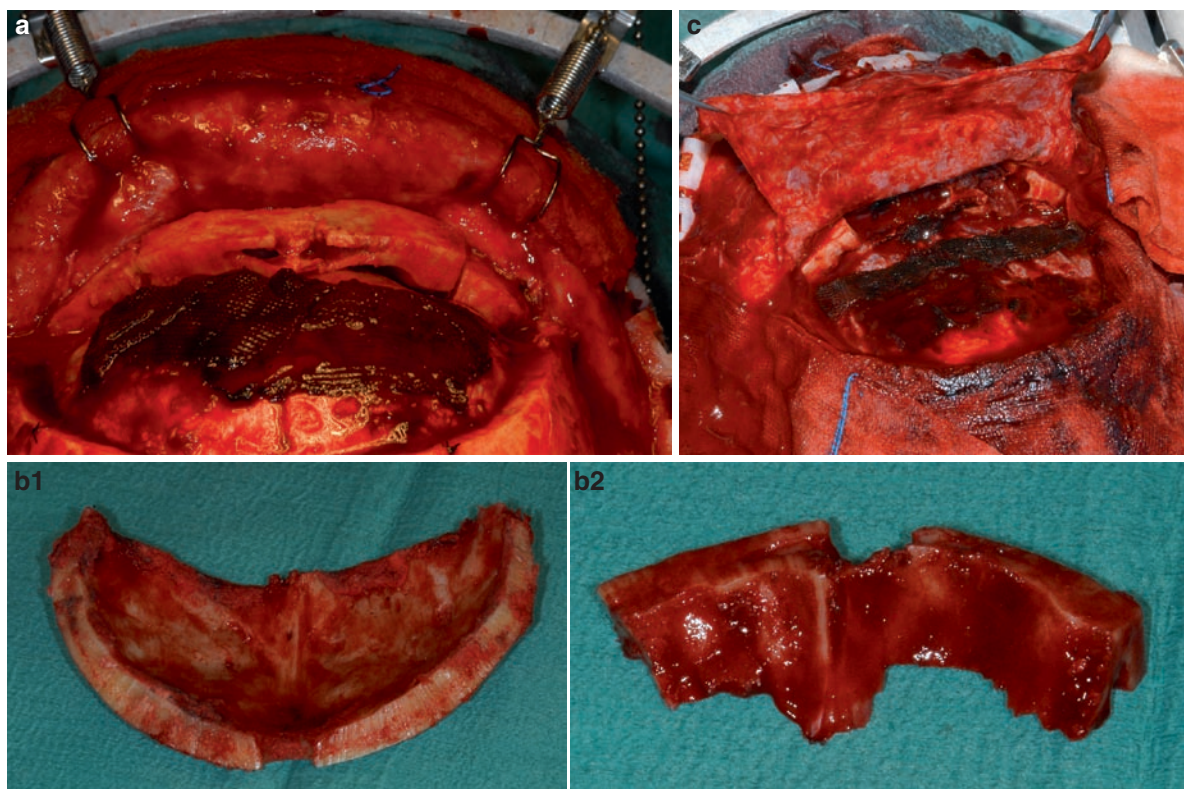


Fig. 8.16 Craniotomy and additional frontofacial osteotomy to facilitate horizontal exposure of the medial frontobasal region. (a) Intraoperative view after bifrontal craniotomy and frontofa-

cial osteotomies. (b) Frontal bone flap and frontofacial segment. (c) Extended horizontal approach to the frontobasal area after removal of the frontofacial segment. Pericranium flap reflected

The dura often rips when removing it from the ethmoid/nasal roof, as it is very thin but strongly adherent in this area (Imhof 2000). The olfactory fibers must be resected in order to expose posterior fractures.

Bleeding from small dural vessels is carefully controlled by applying H_2O_2 cotton pads, with the bipolar Malis forceps and a temporary coverage of minor bleeding spots with a thin layer of resorbable collagen fleece. Dural tack-up sutures are essential as hemorrhage prophylaxis (Figs. 8.18 and 8.19).

After complete exposure of the basal area, the frontal lobe of the brain is carefully retracted, so providing an unhindered view of the posterior wall of the frontal sinus, orbital roof and the anterior skull base.

Due to intraoperative manipulation of the anterior skull base, there may be hemorrhage from the frontobasal dura. This can be stopped by coagulation and the application of adrenaline-soaked pads, so omitting further neurosurgical measures (Imhof 2000).

For protection, the frontal lobe of the brain is covered with a moist membrane (Merocel) and retracted with flexible brain spatulas. Excessive pressure to the frontal lobe can rapidly result in secondary cerebral edema with respective swelling and thus impede or even inhibit its exposure.

In frontobasal fractures, small nondislocated bony fragments are, if possible, left in the position. Larger fragments are sometimes difficult to mobilize and reduce, without injuring vessels or neural structures. Large, loose fragments are placed into antibiotic solution and later used to reconstruct the osseous skull base. If defects of the frontal base result, autogenous bone grafts have to be used for reconstruction (Imhof 2000).

- Intradural exposition of the skull base

If frontobasal fractures are combined with cerebral injuries which need revision or if extensive frontobasal fractures with dural tears lie in the vicinity of the nasal roof

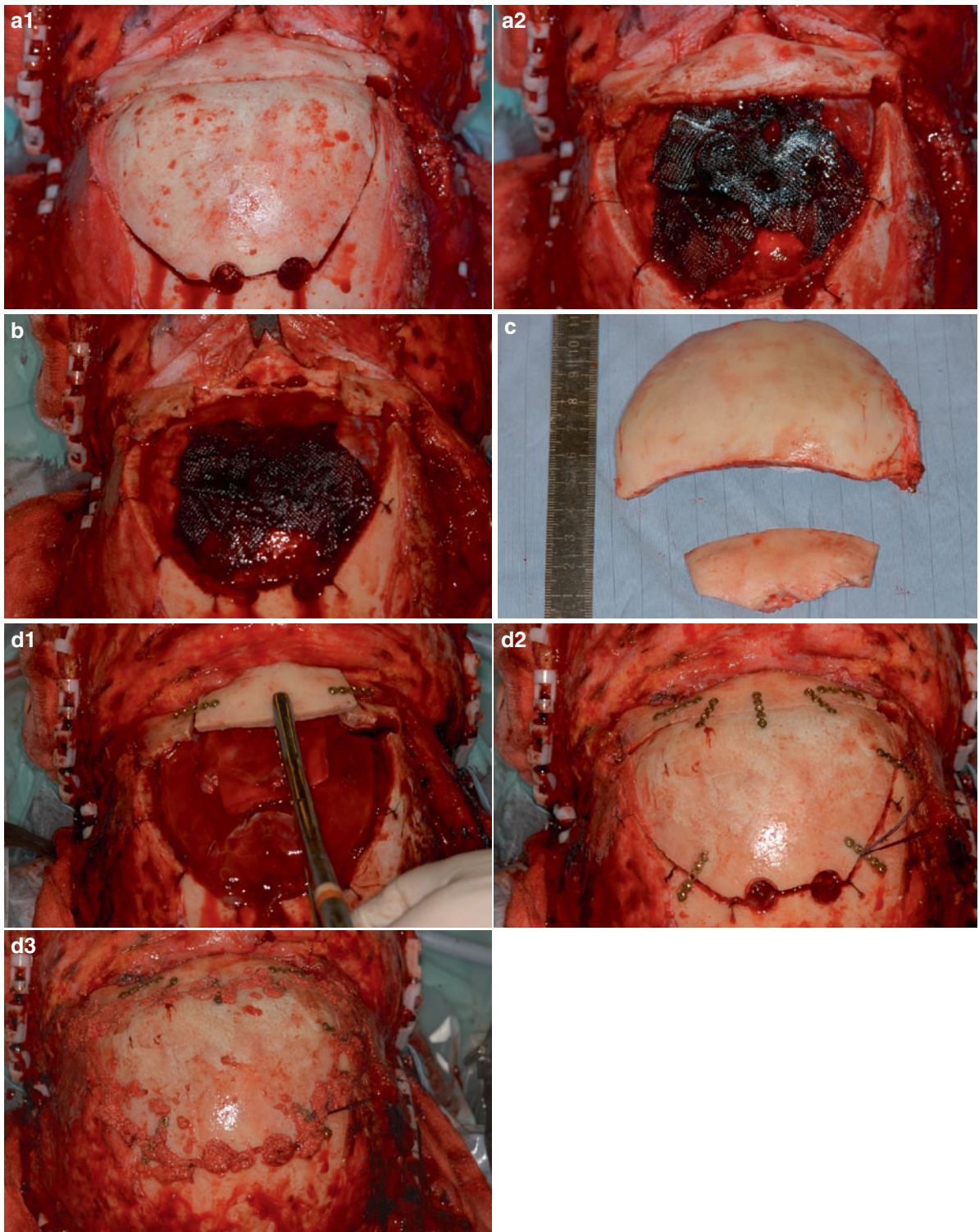


Fig. 8.17 Craniotomy with additional frontofacial osteotomy. (a) Bifrontal osteotomy. (b) After removal of the frontofacial segment excellent exposure of the frontobasal area is achieved without extensive brain retraction. (c) The frontal flap and the

frontofacial segment. (d) Reintegration and fixation of the frontofacial segment and the bone flap. Burr holes and bony gaps are filled with bone dust

or cribriform plate and the ethmoidal cells, then both the transfrontal *extradural and intradural access* are used.

Neurosurgical indications for the intradural approach (Schmidek and Sweet 1988) are:

- Intracranial hemorrhages.
- Impalement injuries with lesions of the brain tissue.
- Complicated craniofacial fractures with severe cranio-cerebral trauma, ramified dural tears and profound cerebral injuries.
- Treatment of skull base lesions, dural injuries and cerebral wounds, in extreme posterior positions, whereby the border is denoted by the anterior ethmoidal artery. The region dorsal dorsal to this vessel corresponds with the posterior ethmoid.
- Large dislocated depressed fractures of the frontal bone.

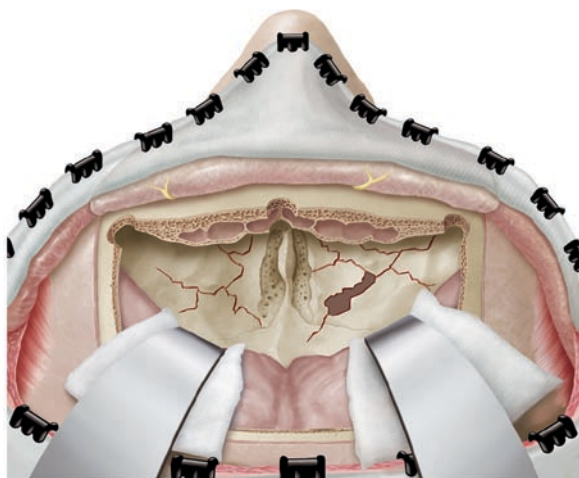


Fig. 8.18 Schematic diagram of the transfrontal extradural approach to the frontobasal area with exposure of the posterior basal fractures after sacrificing of the olfactory nerves (mod. a. Samii et al. 1989; Weerden 1995)

- Unclear localization of liquor fistulae. A common situation, in which the intradural approach is of advantage because of its clear view.
- In situations in which fistula closure presents the main problem, the intradural approach is more reliable than the extradural (Probst 1986). This is particularly relevant in cases of secondary intervention (delayed treatment). The olfactory fibers can be preserved with the intradural approach.

Technical Aspects

With the intradural approach the frontal skull base can be reached from a medial, fronto-lateral, or interhemispherical access, depending on the location of the dural defect (Imhof 2000).

Following dural incision and division of the superior sagittal sinus, the frontal lobe is raised with a cerebral spatula and the frontal base presented in full view. Potential contusion hematomas and regions of cerebral necrosis, which might obliterate the basal dural defect and so mask a rhinoliquorrhea, can be removed (Ewers et al. 1995).

After temporarily covering the outer cerebral surface, bone fragments from the orbital roof and the anterior cranial fossa are repositioned to prevent enophthalmus (Figs. 8.20 and 8.21).

- The intradural access provides a good view of the dura in the frontal skull base and cerebral fossa. This approach is, however, not well adapted for acute treatment following severe cranio-cerebral trauma as the risk of further cerebral injury exists during the inevitable retraction of the frontal lobe (Lehmann et al. 1998).

Fig. 8.19 Transfrontal extradural exposition of the anterior frontal base presenting the basal fractures



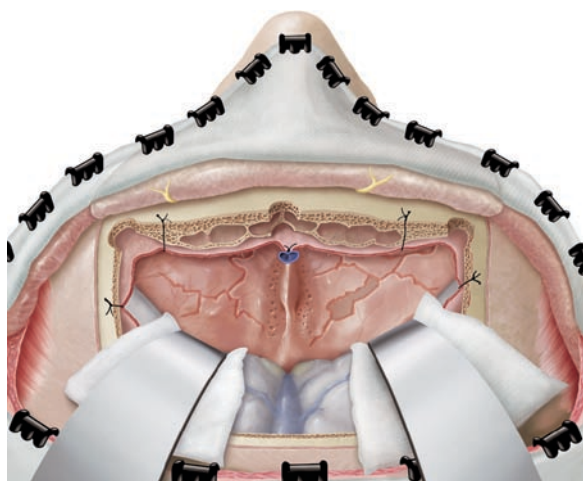


Fig. 8.20 Schematic diagram of the transfrontal intradural approach to the frontobasal area and visualization of the injured dura (mod. a. Samii et al. 1989; Weerden 1995)

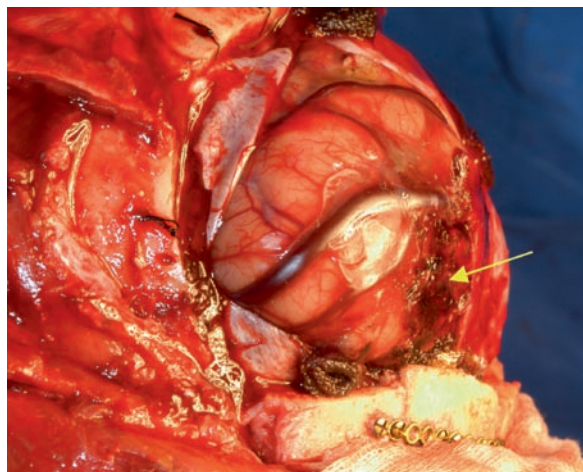


Fig. 8.21 Transfrontal intradural approach to the frontobasal area. Intraoperative view demonstrating intradural exposure of the traumatized right frontal lobe (arrow)

8.4.3 Advantages, Disadvantages, and Risks Associated with the Transcranial Approach

Without doubt the extradural and intradural approaches pose a greater operative risk than the subcranial approach, particularly when early surgery is undertaken. The danger of postoperative cerebral edema is far greater in recent cerebral injuries (Probst and Tomaschett 1990). With approximately 3% there is a much lower mortality rate after the edema has subsided and the cerebral parameters have normalized (Loew et al. 1984).

Postponed intracranial intervention in the treatment of dural and skull base lesions, awaiting the subsidence of the cerebral edema, consequently results in a postponed primary surgical treatment of the midfacial fractures and the skull base by 2-3 weeks. As a result of delayed surgery and beginning fracture consolidation, bone reduction is complicated and hence may entail secondary surgery at a later date.

Furthermore an *increased risk of ascending infection* exists from deferred surgery owing to congestion and secretion in the paranasal sinuses as well as an increased risk of meningitis resulting from the untreated skull base fractures (Vuillemin et al. 1988).

A *partial or total anosmia* is not always avoidable. This may either be a consequence of trauma, particularly from dislocated fragments in the medial skull base region, or it may result from surgical exploration, especially if the olfactory fibers have to be transected bilaterally (Samii 1989). Olfactory dysfunction (hyp- or anosmia) as a result of the surgical transcranial intervention was found in 7-8% (Neidhardt 2002; Schroth et al. 2004).

Other disadvantages may arise from the comparatively large *wound surface* and technically from the problematic treatment of the olfactory groove, concerning preservation of olfactory function, the direct contact with the cerebral cortex, pontine veins and the superior sagittal sinus (Füssler et al. 1996; Rosahl et al. 1996; Lehmann et al. 1998).

The narrow access to the sphenoidal sinus and further dural tears as a consequence of surgery in areas where it is particularly adherent (medial ethmoidal roof, cribriform plate, crista galli) pose additional risks (Lehmann et al. 1998).

With a supplementary frontofacial osteotomy the frontal base can be tangentially exposed, bone fragments removed and the skull base reconstructed without excessive retraction of the cerebral structures. The surgical trauma is minimized and at the same time a radical ethmoidectomy – as in the subcranial approach – can be avoided (Kuttenberger and Hardt 2001).

8.5 Transfrontal-Subcranial Approach

The classical extracranial-transethmoidal approach is limited to the frontal sinuses and the ethmoid-sphenoidal region and is therefore not an adequate alternative to the neurosurgical approach (Samii and Draf 1978; Calcaterra 1980, 1985; Elies 1982; Loew

et al. 1984; Myers and Sataloff 1984; Strohecker 1984; Probst 1986).

Raveh et al. (1984) and Raveh and Vuillemin (1988, 1992) further developed the transthemoidal method and converted it to a subcranial approach, through which one can work subcranially parallel to the skull base level, so avoiding excessive iatrogenic trauma of the cerebral structures. This is especially important where cerebral edema is already present (Raveh and Vuillemin 1992; Raveh et al. 1993; Donald 1994; Lehmann et al. 1998; Fliss et al. 1999).

8.5.1 Indications

Based on the most frequent fracture variations in the region of the anterior cranial fossa, the subcranial approach enables the treatment and reconstruction of the frontal skull base structures along the median and medio-lateral regions of the anterior cranial fossa (Moore et al. 1999) (Fig. 8.22).

- In the majority of cases, the indications for a subcranial approach are limited to localized injuries of the peri- and interorbital regions (NOE fractures) with circumscribed median frontobasal fractures without intracranial injuries (Kellman 1998; Lädach and Raveh 2000).

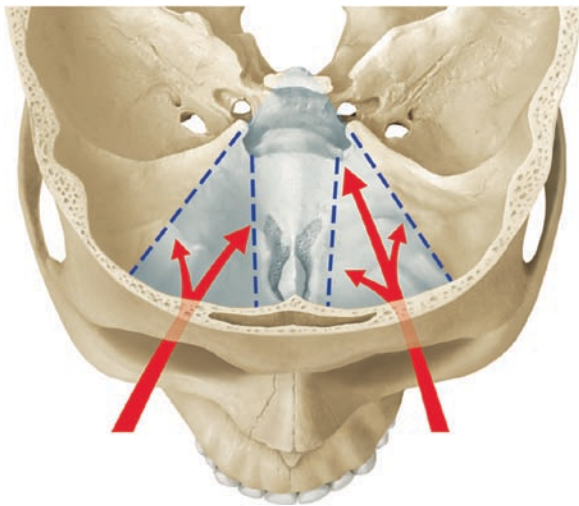


Fig. 8.22 Schematic diagram demonstrating the extent of exposure along the skull base which is possible through the extended subcranial approach (mod. a. Vuillemin et al. 1988; Lädach et al. 1999; Lädach and Raveh 2000)

8.5.2 Surgical Principle

Following a transfrontal coronal approach, resection of fronto-nasal bone fragments and a selective craniotomy, the ethmoidal cells are radically cleared out and the entire median subbasal region, the medial orbital walls, medial orbital roof and orbital apex exposed from a subcranial aspect (Raveh and Vuillemin 1988, 1992; Lädach et al. 1995, 1999; Gliklich and Lazor 1995; Raveh and Lädach 1997; Kellman 1998; Raveh et al. 1998; Gliklich and Cheney 1998; Moore et al. 1999).

8.5.3 Subcranial Surgical Technique

The subcranial approach to the skull base is carried out by temporary resection of the *naso-fronto-cranial fragments*, and occasionally to obtain a better view, an additional selective osteotomy in the naso-frontal region is performed with preservation of the osteotomized segments (Raveh and Vuillemin 1988).

The size of the collaterally removed segments depends on the extent of the fracture site and involves either only the narrow fronto-nasal or additionally the cranio-fronto-nasal region (Vuillemin et al. 1988; Raveh and Vuillemin 1992; Raveh et al. 1998; Lädach et al. 1999; Lädach and Raveh 2000).

Subsequently, after removing the fracture fragments and an additional fronto-nasal segment, a subbasal debridement of the ethmoidal cells is carried out with a *radical ethmoidectomy and total removal of the ethmoidal mucosa*, so exposing the fractured frontal base, including the sphenoid region and the medial orbital walls (Raveh and Vuillemin 1988; Vuillemin et al. 1988).

Due to the enhanced access through the transthemoidal-extracranial approach, the medial aspects of the orbital roof and the orbital apex become visible, allowing a *decompression of the optic nerve*. In such cases, the medial wall of the optic canal is removed transthemoidally and the region is drained to prevent a postoperative apex syndrome (Lädach et al. 1995; Lädach and Raveh 2000). Following dural treatment, reconstruction of the frontobasal structures is performed along the median skull base and the medio-lateral region of the anterior cranial fossa with reconstruction of the orbital roof.

Definite dural sealing is accomplished by using autogenous fascia-lata grafts (Raveh et al. 1998). Subsequently, the extracted fronto-cranial segments are

reintegrated and fixed to reconstruct the osseous frontal region. Simultaneously, the midface fractures are repositioned and fixed with titanium miniplates. An existing pseudo-hypertelorism is corrected.

Subcranial approach to the upper craniofacial and frontobasal fractures (Vuillemin et al. 1988; Ldrach et al. 1999).

- Subcranial approach through coronal incision, temporary removal of the fronto-nasal fragments, selective craniotomy, subsequent ethmoidectomy and revision of the frontal base
- Osseous frontobasal reconstruction, dural repair with fascia lata and reconstruction of the anterior wall of the frontal sinus

8.5.4 Advantages, Disadvantages, and Risks Associated with the Subcranial Approach

The major advantage of the subcranial-transethmoidal approach is avoiding the retraction of the frontal cerebral lobe with potential collateral edema formation. Subdural exploration also reduces the risk of lesions of the olfactory fibers (Raveh and Vuillemin 1988). Thus, the subcranial approach offers an important alternative to the traditional neurosurgical approach to the frontal skull base (Vuillemin et al. 1988; Raveh et al. 1993). The smaller wound and superior evaluation of the olfactory nerve groove speak in favor of the subcranial approach where indicated (Lehmann et al. 1998).

The danger of *skull base instability* exists (Samii 1987, 1989) in cases of extensive debridement when the subcranial transethmoidal approach is used. Furthermore, the limited exposure offered by the subcranial approach is not free of risks in the strongly traumatized midface. Fractures in the region of the lateral and central orbital roofs cannot be reliably inspected and treated.

The access to the lateral sections of the anterior cranial fossa is strongly limited. Injuries to the orbital walls with orbital laceration and formation of orbital hematomas endangering the optic nerve have been described (Hosemann et al. 1991; Rohrich and Hollier 1992).

Loss of osseous structures from the medial orbital walls may lead to *prolapse of the orbital contents* into the interorbital space and, thus, to increase of the orbital

volume with loss of muscular balance, disturbed eye motility, enophthalmus, and infection.

It may, therefore, be necessary to reconstruct the orbital walls during the primary treatment as long as there is no polyfragmentation. A secondary reconstruction of residual, medial defects is technically challenging with inferior results.

Unnoticed *dural tears* may appear as an iatrogenic consequence of a radical ethmoidectomy and basal debridement with possible danger of recurrent liquorrhea.

The subcranial approach only allows modified treatment of cerebral injuries and multiple dural tears (Probst 1986).

Further disadvantages of the subcranial approach are the limited possibility to adequately treat accessory *neurosurgical injuries*, such as accompanying sub- and epidural hematomas and cerebral injuries, which are not accessible from this approach (Samii et al. 1989).

Reconstruction of the orbital roofs with exact fragment placement is more difficult to realize from a subcranial approach than from using the intracranial extradural procedure. Frequently, extensive dislocations of the frontal region, the frontoorbital or frontocranial compartments exist, make an intracranial, extradural procedure inevitable.

Moreover, the healing process of the interorbital bony skeleton with radical removal of all *mucous membranes* has not entirely been cleared (Hosemann et al. 1991).

The outstanding self-healing potential of injured ethmoid cells and the regenerative capacity of the paranasal sinus mucosa, with the recovery of spontaneous ventilation and drainage, brings to question the necessity of a primary radical clearance of the ethmoid cells to access the skull base (Hosemann et al. 1991; Kessler and Hardt 1998a, b).

8.6 Transfacial Approach

The transfacial-frontoethmoidal approach allows clear and sufficient extradural exploration of the frontal sinus, the ethmoid region and the sphenoid sinus as well as the medial orbit; however, with limited access to the medial orbital roof and orbital content.

It offers sufficient space to cover dural defects and placement of drains (Dieckmann and Hackmann 1977; Calcaterra 1985; Ewers et al. 1995; Lange et al. 1995; Schwab 1995; Donald 1998; Schroth et al. 1998).

Using this approach, debridement in the region of the paranasal sinuses and frontal base can be carried out efficiently with microscopic support. This is particularly the case when dealing with fractures in the regions of the cribriform plate, ethmoid roof and sphenoid (Pirsig and Treeck 1977; Draf 1995).

8.6.1 Indications

The indications are in particular localized *skull base fractures* of types Escher II, III, and Oberascher I, IIa. Fractures in the region of the anterior and middle ethmoid radiating into the frontal sinus can be managed *transfacially*. This also applies to fractures of the frontal sinus wall in case of limited bony defects in the posterior wall. Due to the narrow anatomical position, fractures of the posterior section of the ethmoid are difficult to approach transfacially (Oberascher IIb) and are, therefore, preferably treated using the *endonasal* approach. Dural injuries with liquorrhea in the region of the *sphenoid sinus* (Oberascher III), often in combination with uni- and bilateral fractures of the petrous part of the temporal bone, should be treated endonasally using endoscopic techniques.

Extensive craniofacial or fronto-cranial injuries with ethmoid- and frontal sinus fractures in combination with complex midfacial fractures, which necessitate the transfrontal or subcranial-extradural approach in order to reconstruct the facial architecture, are not suited for this approach (Prein 1998).

- Fractures in which lesions of the paranasal sinuses are predominant — as opposed to dura or cerebral injuries — are generally suited for the transfacial treatment; especially those in which facial soft tissue injuries offer a possibility of access (Theissing 1996; Schwab 1995).

8.6.2 Surgical Principle

There are *differing concepts* in treating ethmoidal cell fractures with and without skull base involvement:

Primary radical clearance of the ethmoid cells is an alternative to the *conservative-surgical procedure*. Both variations have a high priority to reestablish ventilation and drainage of the paranasal sinuses.

Up until the late 1970s, literature references recommended radical clearance of injured naso-ethmoidal cells, even without verified dural injuries. More recent publications (Theissing 1996) only speak of a relative indication for debridement in strongly dislocated ethmoid cell fractures. Experimental and clinical research by Hosemann et al. (1991, 1993) has had fundamental consequences on the treatment of naso-ethmoidal fractures.

According to anatomical differences, Hosemann et al. (1991) differentiated between the therapy for *anterior and posterior ethmoid cells*. Fractures of the *posterior ethmoid cell region* generally legitimate an expectative conservative treatment. In the *anterior ethmoid cell region* there is only a relative indication for radical clearance.

Hosemann et al. (1991) stress the significant self-healing potential and regeneration ability of the ethmoidal cell mucosa with recovery of spontaneous ventilation and drainage of the ethmoidal system, even with extensive fracture comminution. Regeneration of the ethmoidal cell system even in strongly dislocated and comminuted fractures of the interorbital complex were demonstrated by means of a retrospective study based on CT scans (Kessler and Hardt 1998a, b).

A comparison of craniofacial fractures treated with cranial ethmoid cell debridement with subcranial midfacial and NOE fractures treated without ethmoid cell debridement showed no relevant postoperative differences in the subsequent long-term CT follow-up (Kessler and Hardt 1998a, b).

- These results question the necessity of primary radical ethmoidal clearance in comminuted anterior skull base fractures (Hosemann et al. 1991, 1992, 1993; Kessler and Hardt 1998a, b) (Figs. 8.23 and 8.24)

8.6.3 Transfacial Surgical Approaches (Denecke et al. 1992; Schwab 1995; Theissing 1996; Ernst et al. 2004)

For a unilateral transfacial approach the paranasal semicircular incision according to Kilian and for a bilateral approach the butterfly-incision according to Siebenmann are used (Ewers et al. 1995; Boenninghaus 1971, 1974).

As is often the case in frontobasal injuries, there are facial soft tissue injuries which make it necessary to

Fig. 8.23 Course of a craniofacial fracture with cranial ethmoid cell debridement (a. Kessler and Hardt 1998a, b). (a) Axial CT of a craniofacial fracture with complete demolition of the ethmoid cell architecture following gun shot injury. Loss of the left eye (Wassmund III right, Le Fort II left, NOE fracture, zygoma fracture left). (b) CT control 22 months postoperatively. Architecture, ventilation, and drainage of the ethmoid cells are restored

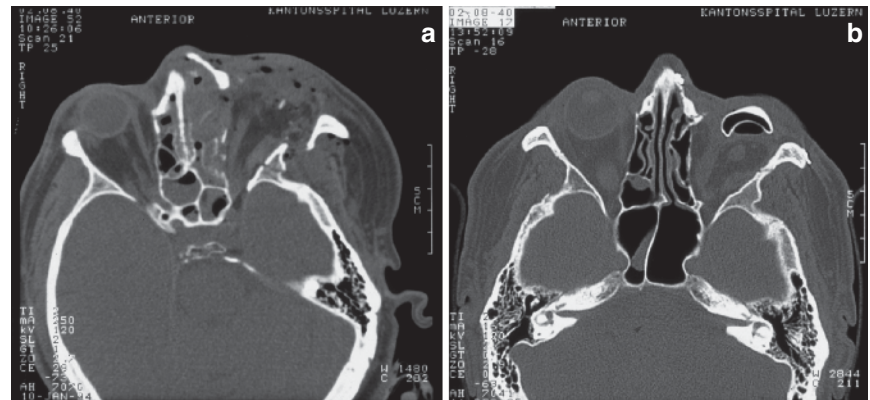
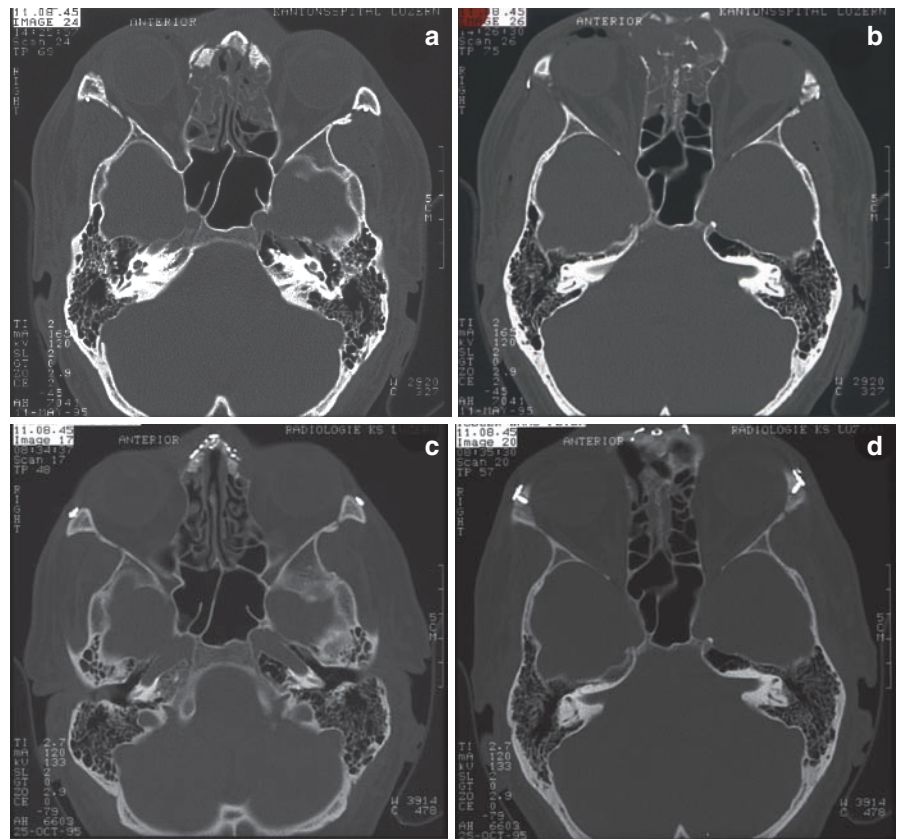


Fig. 8.24 Course of a subcranial fracture without ethmoid cell debridement (a. Kessler and Hardt 1998a, b). (a, b) Axial CT of a central midfacial comminuted fracture with demolition of the NOE complex. (c, d) CT control 6 months postoperatively in the same plane. Anatomically acceptable restitution of the ethmoidal cell system



modify the classical approaches (Dieckmann and Hackmann 1977).

If the butterfly incision is continued laterally via the zygomatic bone, a cranially based bipedicle flap can be created and used in craniotomies for treating skull base fractures with the intra- and extradural approach (Ewers et al. 1995).

1. The semicircular incision takes its course below the inner border of the eyebrow, continues caudally between the bridge of the nose and the medial corner of the eye and ends at the height of the inferior orbital margin (Schwab 1995). The incision may also be combined with a primary Z-plasty in the corner of the eye.

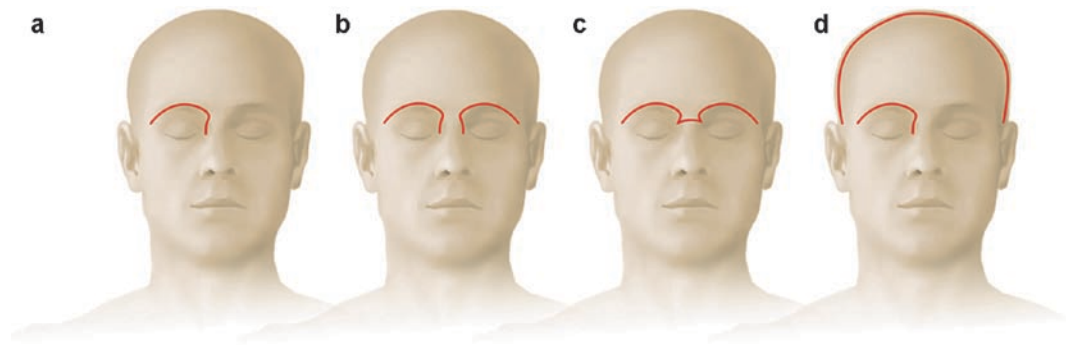


Fig. 8.25 Different transfacial approaches for exposure and revision of the frontal sinus, ethmoid, orbit, and sphenoid (mod. a. Probst 1971). (a) Unilateral subbrow incision (frontoorbital

approach after Killian). (b) Bilateral subbrow incision. (c) Butterfly incision connected through a glabellar crease. (d) Coronal incision combined with a median subbrow incision

By strictly holding the scalpel vertically the skin, the subcutaneous tissue and the periosteum are cut simultaneously through to the bone. Bleeding from the supraorbital artery must be carefully controlled. Whilst performing the incision the lacrimal sac and the lacrimal canals must be protected as far as the entrance point of the nasolacrimal canal. Subsequently, the soft tissues and periosteum from the floor of the frontal sinus and lateral slope of the nose are dissected. It is necessary to perform a strictly subperiosteal dissection around the insertion point of the medial palpebral ligament and trochlea. Particular care must be taken to maintain the integrity of the medial palpebral ligament, in order to prevent a secondary hypertelorism. The lacrimal sac is released from its bony groove (Figs. 8.25 and 8.26).

2. Subsequently, the floor of the frontal sinus and the ethmoid can be opened through this approach, either by an osteoplasty or by a bone resection. *Bone trepanation* (lacrimal bone - anterior section of the lamina papyracea) in the medial orbital corner above the lacrimal fossa, provides access to the frontal sinus, ethmoid cells and orbital roof. If the anterior wall of the frontal sinus is comminuted, bony fragments should be removed and reimplanted at the end of the operation if possible. The fragments are preferably fixed with microplates.
3. After *opening* and *clearing* the ethmoidal cells and carefully removing the bony fragments in the region of the posterior wall of the frontal sinus, roof of the ethmoid and cribriform plate, transethmoidal removal of the anterior wall of the sphenoid sinus with exploration of the roof of the sphenoid sinus is possible.
4. Following *exploration of the ethmoidal roof* and the cribriform plate, the osseous defect in the skull base

is enlarged using a diamond or trepan burr under endoscopic control, so providing a good view of the dural defect. Fluorescein sodium administered intrathetically prior to surgery results in an improved intraoperative presentation of dural defects (Theissing 1996; Ernst et al. 2004).

5. In order to sufficiently seal any dural leak, it is essential to have a *complete view of the entire dural tear*. The dura is primarily sutured after it has been carefully lifted from the bone. In order to protect the olfactory fibres as far as possible, no extensive manipulations should be done here. A graft is then placed between bone and dura to cover the defect (Ernst et al. 2004).

In the case of a bilateral fracture, the transplant has to be extended to the opposite side to cover the complete defect. A second graft overlaps the bony defect and is fixed with fibrin glue. The reconstruction should be supported by a nasal tamponade for 7 days (Ernst et al. 2004) (Fig. 8.27).

8.6.4 Dura Treatment in the Frontal Skull Base

8.6.4.1 Frontal Sinus

Posterior wall defects of the frontal sinus are mostly found at the point of transition to the ethmoid.

- *Simple osseous defects without dural injury* are covered with a patch (fascia/alloplastic material) and sealed with fibrin glue. Bony fragments can be replaced in order to reduce the size of larger defects.

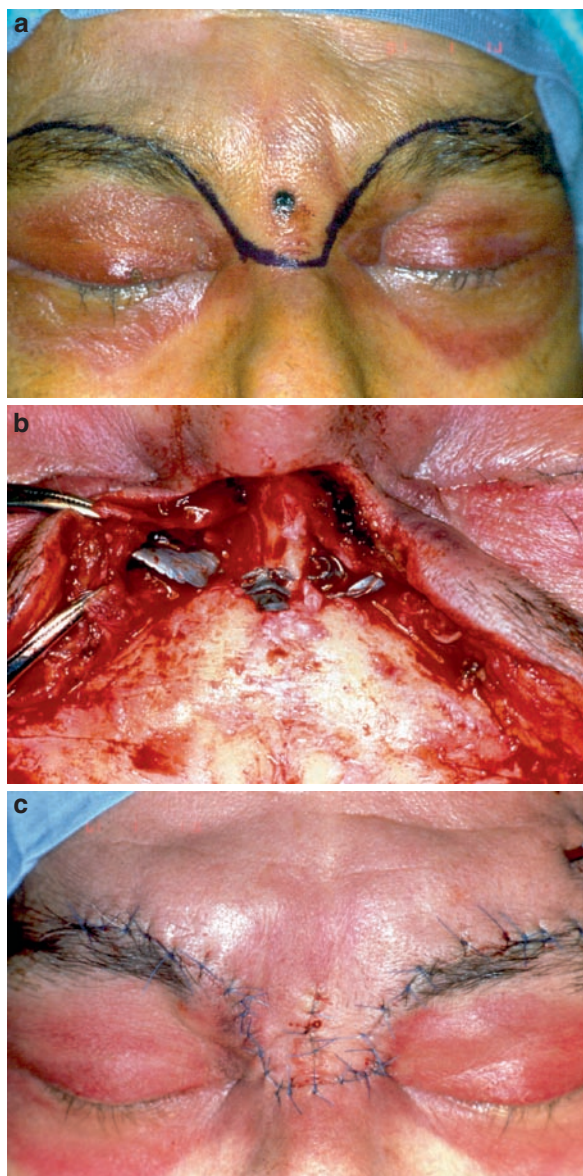


Fig. 8.26 Transfacial butterfly incision to treat a gun-shot injury in the naso-frontal region. (a) Incision outlined. (b) Intraoperative situation demonstrating nasofrontal fracture. (c) Situation after wound closure

- *Dural tears* in the region of the posterior wall of the frontal sinus are easily accessible after sufficient exposure. They can be well repaired by *dural suture* using atraumatic, nonabsorbable sutures, as the dura in this area exhibits the necessary consistency (Schwab 1995). In addition, a *duraplasty* is carried out using alloplastic material or fascia, which is fixated with fibrin glue. A drain is placed into the nasal cavity to

assure frontal sinus ventilation and drainage. This type of drainage should be maintained for 3–6 months until total consolidation and reepithelization has taken place.

8.6.4.2 Ethmoid/Cribriform Plate

The intact dura is dissected from the bony rims of the fracture site and a bed formed for the necessary patch. This is then placed between dura and bone and fixed with tissue adhesive. Free pericranial or fascia lata grafts or alloplastic transplants may be used for extradural duraplasty (Schwab 1995; Ernst et al. 2004).

8.6.4.3 Sphenoid

After ethmoidectomy of the posterior ethmoid, the anterior wall of the sphenoid sinus is removed under microscopic control (Delank et al. 1998). When clearing the fragments from the sphenoid sinus, great care must be taken as there is a potential danger of injuring the local vessels. Defects should be covered with a transplant (Landreneau et al. 1998).

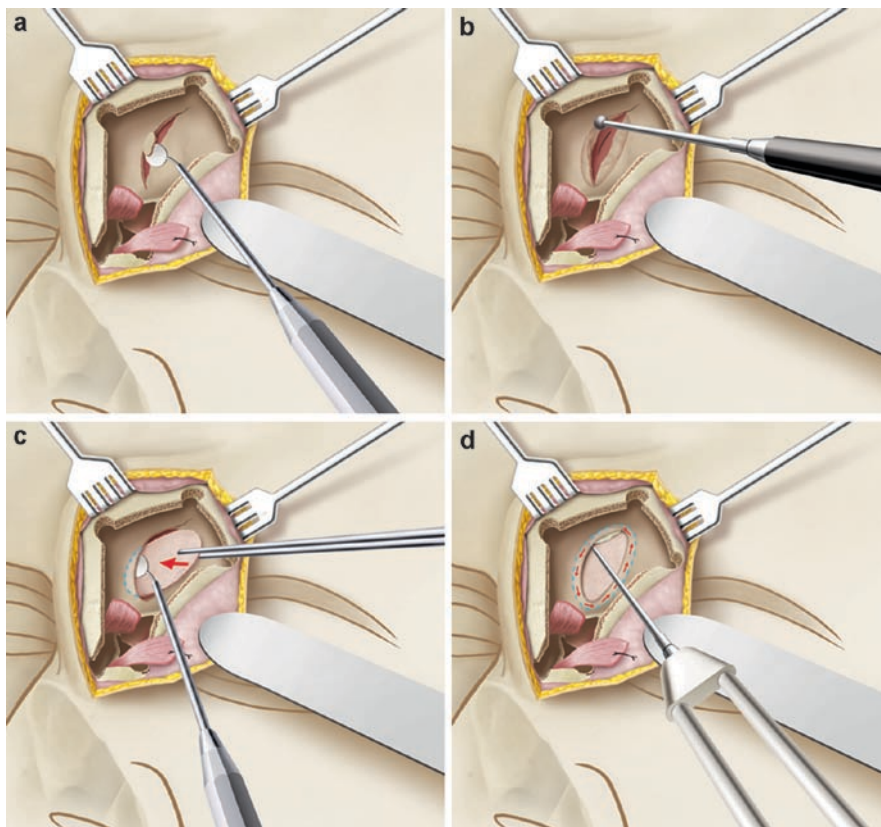
If there is a dural fistula in the *roof of the sphenoid sinus*, the mucous membrane is carefully removed and the entire sphenoid sinus is lined with a transplant, sealed, and filled with fibrin foam. Closure to the nasal cavity is secured and supported by an additional tamponade strip.

The obliteration technique is advocated for *covering large sphenoidal sinus defects*, i.e., the entire sphenoid cavity is completely filled with a transplant. Fat, muscle, or fascia tissues can be used (Schwab 1995; Schick et al. 1996; Delank et al. 1998; Ernst et al. 2004).

8.6.5 Advantages, Disadvantages, and Risks Associated with the Transfacial Approaches

The transfacial butterfly incision can result in an esthetically nonacceptable scar and offers only a limited exposure. Simultaneously with this incision, the supraorbital and supratrochlear nerves may be injured. The butterfly incision has the same limiting factors as the approach through penetrating soft tissue

Fig. 8.27 Treatment of fractures of the posterior wall of the frontal sinus with laceration of the dura (mod. a. Theissing 1996). (a) Osteotomy of the anterior wall of the sinus and removal of comminuted posterior wall fragments. (b) Exposure of the dura by removal of bone with diamond burr. The dura is circular released from the bony margins. (c) Foisting a transplant between dura and posterior wall. (d) Fixation of the graft with fibrin glue



wounds and should only be used in selected cases with marginally displaced fractures of the anterior wall of the frontal sinus.

Risks associated with the transfacial transethmoidal-subdural approach:

- Injury to the orbital walls, orbital hematoma
- Increased orbital volume, enophthalmus
- Risk of injuring the optical nerve
- Iatrogenic dural injuries
- Damage to the olfactory fibers

A significant disadvantage of the transfacial-transethmoidal approach is that *simultaneous cerebral injuries cannot be adequately treated*.

Efficient fistula closure is particularly problematic in regions where the intact and strongly adherent dura cannot easily be released from the bone (ethmoid roof, cribriform plate). The subbasal extradural plasty is less reliable than the intracranial one (Probst 1986; Lange et al. 1995). Bilateral and/or multiple lesions treated extracranially are more difficult to treat because of the poor view. Additionally, the entire orbital roof cannot be inspected or reduced.

Extensive debridement of the ethmoid by multifragmentation of the medial osseous orbital wall may lead to additional instability of the orbit. This is particularly relevant for the so-called *orbital-key area* (sino-ethmoidal angle). This region is particularly important in stabilizing the orbit. If debridement of the osseous orbital frame takes place in this region and the bony stability is reduced, orbital tissue prolapse into the paranasal sinuses may result (Ernst et al. 2004).

8.7 Endonasal-Endoscopic Approach

The endoscopically based nasal surgery for treating base fractures in the anterior cranial fossa is exclusively endonasal (Draf 1978, 1981, 1992; Wigand 1989; Stammberger 1991; Thumfarth et al. 1998; Gjurić et al. 1996; Pia und Aluffi 1997; Burns et al. 1996; Schick et al. 1997, 1998).

The endonasal technique causes the least trauma, but is only reliable in circumscribed skull base fractures with liquor fistulas in the fronto-ethmoidal region.

8.7.1 Ethmoid Roof Fractures: Surgical Principles

Isolated frontobasal fractures of the posterior and mid-ethmoidal sections can be treated from the endonasal-extradural approach. The definitive treatment of ethmoid roof fractures follows that of accompanying midfacial fractures, which should be repositioned and stabilized beforehand.

After the ethmoid cells have been opened, the defect in the skull base is exposed using the diamond burr under endoscopic and microscopic control, so providing a full view of the dural injuries. As the dura is quite adherent in this region, it may be necessary to repress the brain with gauzes. Defect exposure in the region of the crista galli may be difficult. Injured and prolapsed brain tissue is repositioned using protective gauzes; hematomas are evacuated.

Reconstruction is always compromised by increased cerebral pressure. Decreasing cerebral pressure by draining the liquor and supporting the reconstruction with a tamponade minimizes this danger (Ernst et al. 2004).

8.7.2 Sphenoid Fractures

Defects in the region of the posterior ethmoid roof and particularly isolated defects of the sphenoid sinus are treated endonasally (Dietrich et al. 1993; Pia and Aluffi 1997; Landreneau et al. 1998; Schroth et al. 1998). The transnasal surgical closure of sphenoid defects has a success rate of about 90% (Stoll 1993; Schick et al. 1996; Delank et al. 1998). The essential drainage of the paranasal sinuses can be performed simultaneously during this intervention.

8.8 Surgical Approaches/ Own Statistics

In 70% of our own patients with craniofacial fractures we chose the transfrontal-transcranial approach, in 20% the transfrontal-subcranial and in 10% the unilateral transfacial-frontoorbital approach.

In 23% the transfrontal-intradural approach was necessary due to extensive cerebral injuries (Neidhardt

2002). (The number of subcranial approaches increases if osteotomies with resection of a fronto-cranio-nasal-segment are added to the subcranial approaches).

Distribution of surgical approaches in craniofacial fractures (Neidhardt 2002)

Transfrontal-transcranial	70%
Extradural	47%
Intradural	23%
Transfrontal-subcranial	20%
Transfacial-frontoorbital	10%

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9.1 Principles of Dural Reconstruction

The dura mater belongs to the bradytrophic tissues of the body. Its healing process is slow and in the form of scarred connective tissue (Probst 1986; Ernst et al. 2004). As the arachnoid membrane heals considerably faster than the dura mater, a manifest liquorrhea may no longer be traceable. However, as long as the dura wound is not closed, the arachnoidal wound closure does not render sufficient protection against ascending infections from the region of the paranasal sinuses (Süss and Corradini 1984; Probst and Tomaschett 1990; Stammberger and Posawetz 1990).

The aim of dural reconstruction is to achieve a watertight closure of the dural defect by doing a straightforward dural suture or a duraplasty using dural tissue substitutes.

There are principally three possibilities of dural closure (Stammberger and Posawetz 1990; Stammberger 1991):

- Intradural treatment (overlay technique):

Positioning of a transplant on the dural defect between the dura and the brain tissue

- Extradural treatment (underlay technique):

Placing a transplant between bone and dura — the transplant acts as a splint for natural dural closure

- Sandwich method:

Intracranial and endonasal coverage of the dural defect (sandwich method used in nasal surgery)

Small and adaptable dural tears are closed extradurally using nonabsorbable sutures. The defects are covered by an additional autogenous transplant. Multiple dural tears or dural defects are covered with autogenous or allogenic or alloplastic transplants using continuous sutures and fibrin glue.

9.2 Dural Substitutes

As there is no or only little primary dural healing following injury, it is essential to position transplants as matrix for a liquor-proof defect closure. The graft's or transplant's connective tissue components lead to scar formation (Probst 1986). Various procedures, depending on defect size, are recommended for dural closure (Schmidek and Sweet 1988; Schick et al. 1997; Rosahl 1999; Ernst et al. 2004):

- Autogenous grafts

Temporal fascia grafts, galea-periosteum grafts, fascia lata grafts, muscle grafts

- Allogenic transplants

Lyophilized transplants (human lyophilized dura, collagen membranes)

- Alloplastic synthetic dural substitutes

Polyurethane-implants/biosynthetic cellulose

9.2.1 Autogenous Grafts

9.2.1.1 Autogenous Fascial Grafts (Stoll 1993; Jones and Becker 2001; Ernst et al. 2004)

The advantage of autogenous implants is that the risk of transmitting diseases — e.g., slow-virus-infections — or the danger of tissue rejection is eliminated. Graft harvesting, however, increases the surgical trauma and morbidity for the patient and lengthens the operations.

Grafts from the fascial sheaths of the rectus femoris muscle (fascia lata), the rectus abdominis muscle or the temporalis muscle are favored. These grafts have a

distinct tendency to shrink and must, therefore, be of sufficient size on insertion (Probst 1986). The grafts are fixed with fibrin glue and sutures.

There is a tremendous amount of clinical and experimental knowledge about autogenous fascia transplantation. All results indicate that free autogenous fascial tissue grafts (fascia lata, temporalis fascia) remain vital (Probst 1971, 1986). Blood supply is assured by direct spontaneous anastomosis between the vessels of the graft and those of the local tissues.

9.2.1.2 Autogenous Muscle Grafts (Probst 1986; Stoll 1993)

In contrast to fascia grafts, autogenous muscle grafts taken from the frontalis, temporalis, or quadriceps femoris muscles share the disadvantageous tendency to shrink and cicatrize and are afflicted with the problems of other free transplants. Similar to autogenous bone grafts, autogenous muscle grafts must be surrounded by vital tissue in order to heal (Stoll 1993) (Fig. 9.1).

9.2.1.3 Pericranium Flap/Graft (Kessel et al. 1971; Jackson et al. 1986; Imhof 2000)

The vital and vascularized pericranial flap is available in a variety of sizes and because of its adaptability and vitality has become standard in skull base reconstruction.

Adjacent sections of pericranium may be used as non-vascularized tissue patches (Price et al. 1988).

9.2.2 Allogeneic Transplants

9.2.2.1 Lyophilized Dura

Freeze-dried lyophilized human dura, which was frequently used in the past, is only rarely used today due to the risk of slow virus infection [Jakob-Creutzfeld disease, bovine spongiform encephalopathy (Martinez-Lage et al. 1993; Lane et al. 1994; Christmann 2003)].

9.2.2.2 Collagenous Compounds

Today there is an increased availability of products based on collagen structural proteins. These compounds are derived from both humans and animals. Chemical processing can alter the connective tissue structures to such an extent that a mere acellular and antigen-free collagen frame remains (Chaplin et al. 1999). As a result, membranes exist which are exclusively composed of collagen fibrils or collagen-laminated substrates — e.g., polyglactin — without bearing the relatively high infectious risks of nontreated allo- or xenogenic transplants (San-Galli et al. 1992).

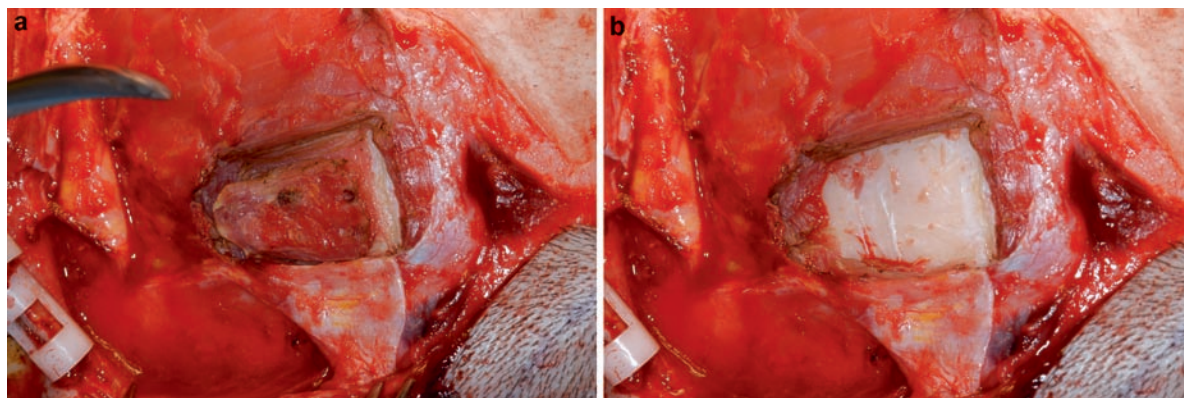


Fig. 9.1 Harvesting of temporal muscle patches. (a) Reflected temporal fascia, muscle patch incised. (b) Muscle patch harvested prior to closure of temporal fascia

The collagen fiber network functions as a matrix, which the connective tissue cells invade and ultimately transform to a neo-dura (Chaplin et al. 1999; Christmann 2003).

9.2.3 Alloplastic Synthetic Dural Substitutes

Dural substitutes comprise a diversity of synthetic materials, among them polytetrafluoroethylene (ePTFE-GORETEX), biosynthetic cellulose and polyesterurethane (NEUROPATCH).

Neuropatch

Neuropatch is a fine fibrillate microporous fleece manufactured from ultrapure aliphatic polyurethane. Its structure is characterized by open micropores on the surface of the fleece, which significantly facilitate an efficient immigration of cells. It is also characterized by exceptional tissue compatibility and biostability.

Fibroblasts migrate into the microporous structure and deposit collagen, so anchoring the fleece to the tissues. There is an absence of giant cell foreign body infiltration. There is no aggregation of lymphocytes, eosinophils, and neutrophil granulocytes or mononuclear macrophages within the implant, which could suggest a chronic inflammatory or hyperallergic reaction. Neomembranes are formed, which encase the dural substitute-fleece. These neomembranes remain very thin and do not form material-induced adhesions with the brain.

The appropriately trimmed fleece should be fixed with nonabsorbable sutures (polyester, polypropylene). Atraumatic sutures enable fixation without damaging the fleece; additionally it is sealed with fibrin glue.

9.3 Principles of Skull Base Reconstruction

Before the skull base is reconstructed, a debridement of the traumatized paranasal sinuses (ethmoid cells, frontal sinus) has to be done in order to prevent ascending intracranial infections. Recommendations range from radical transethmoidal or subcranial-transethmoidal debridement to conservative ethmoidectomy from

a transcranial, extradural approach (Draf and Samii 1983; Probst 1986; Probst and Tomaschett 1990).

9.3.1 Debridement of the Ethmoid Cells

According to neurosurgical experience, removal of debris in the region of the paranasal sinuses can be reliably achieved from the cranial aspect using the surgical technique from Unterberger (1959) (Probst and Tomaschett 1990).

In particular, debridement of the *frontal sinus, anterior, and middle ethmoid* can be carried out efficiently starting in the extradural space.

Debridement of the anterior and middle ethmoid from the cranial aspect provides sufficient communication to the nose to ensure secretion drainage from the posterior ethmoid and the sphenoid sinus into the nasopharynx (Probst and Tomaschett 1990).

If fractures without significant comminution exist, the treatment may be alternatively carried out from a frontoorbital or endonasal approach without ethmoidectomy through the cranial approach. A similar treatment can be used, if the posterior ethmoid and the sphenoid sinus are involved (Imhof 2000; Ernst et al. 2004).

The extradural restoration of the posterior ethmoid cells and the sphenoid sinus, however, is problematic. Following severe injury, the subarachnoidal space often cannot be identified and the comminuted paranasal sinuses are communicating with the subarachnoidal space (Probst and Tomaschett 1990).

If the basal dura over the posterior ethmoid cells (arachnoidal and dural tears) and over the sphenoid sinus is severely injured, it has to be repaired by a combined endonasal (ENT) and intradural approach (NC) (Ernst et al. 2004).

9.3.2 Debridement (Cranialization) of the Frontal Sinus

Whenever possible, the anterior sinus wall is preserved or reconstructed when performing an osseous debridement in the region of the frontal sinus (Ewers et al. 1995).

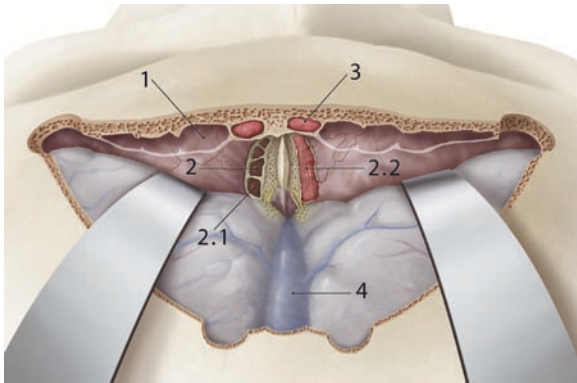


Fig. 9.2 Exposure of the frontal skull base after cranialisation of the frontal sinus, obliteration of the nasofrontal duct and debridement of the anterior and middle ethmoidal cells (mod. a. Imhof 2000). 1 Frontal sinus (after cranialization), 2 ethmoid cells (2.1 post-ethmoidectomy, 2.2 closure with muscle patch and fibrin sealant), 3 nasofrontal duct (obliteration with muscle patch and fibrin sealant), 4 superior sagittal sinus

In comminuted fractures of the anterior and posterior walls of the frontal sinus or extensive damage to the posterior wall of the sinus, reconstruction of the anterior wall and resection of the entire osseous posterior wall, including meticulous removal of the entire mucous membrane, is carried out with a diamond trephine. Inverted closure of the naso-frontal duct avoids postoperative complications of ascending infections (Fig. 9.2).

9.3.3 Skull Base Repair

9.3.3.1 Extradural Skull Base Repair

- Muscle/fascia patch

Following debridement of bony fragments and the loose parts of the mucous membrane in the region of the ethmoid cells, all osseous defects of the skull base are routinely covered with an autogenous free muscle or fascial graft (Draf und Samii 1977; Probst 1986; Derome 1988; Sundaresan and Shah 1988; Probst and Tomaschett 1990; Ewers et al. 1995).

Small fronto-ethmoidal defects are covered with a fascia- or neuro-patch and a pedicled, pericranial flap.

In larger defects — particularly following ethmoid cell debridement — the basal defect coverage initially consists of one to two layers of hemostyptic gauze (e.g., Tabotamp), followed by an autogenous muscle patch,

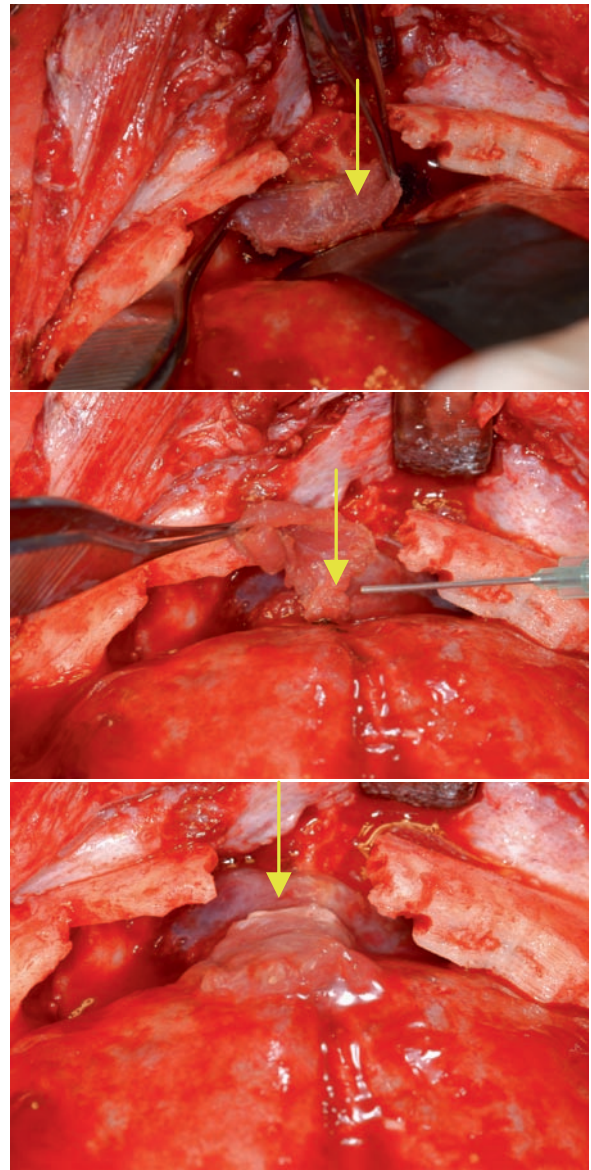


Fig. 9.3 Closure of the medial anterior skull base area: obliteration of the nasofrontal duct with muscle patch and fibrin sealant (arrow) after cranialisation of the frontal sinus and ethmoidectomy

which is fixed with fibrin glue and then covered with a pedicled pericranial flap (Probst 1986) (Fig. 9.3).

Closure of the nasofrontal ducts after cranialization of the frontal sinus is obligatory. Their mucous membranes are circularly peeled off, pushed downwards, invaginated, and sealed with fibrin glue. Final coverage of the duct and floor of the frontal sinus is achieved in the same way as described above using fibrin glue, a muscle patch and optionally a pericranial flap (Stanley and Schwartz 1989) (Fig. 9.4).

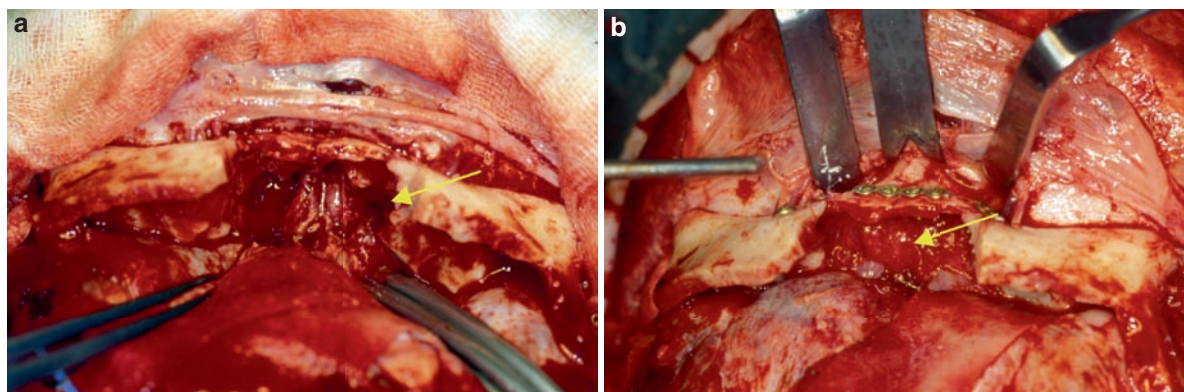


Fig. 9.4 Closure of the frontal skull base. (a) Intraoperative view after median nasofrontal osteotomy and cranial ethmoid-debridement (arrow). (b) Obliteration of nasofrontal duct with autogenous muscle patch (arrow)

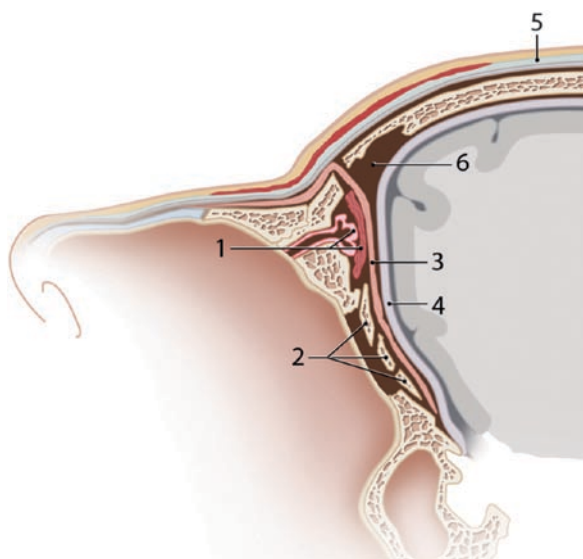


Fig. 9.5 Schematic diagram illustrating extradural treatment of fractures of the frontal skull base. Cranialization of the frontal sinus, obliteration of the nasofrontal duct, closure of the skull base defects with bone grafts and pericranial flap. The pericranial flap is inserted through a slot beneath the inferior rim of the frontal bone flap (mod. a. Imhof 2000). 1 Obliterated nasofrontal duct with muscle patch, 2 bone fragments/bone grafts/chips, 3 inserted pericranial flap, 4 dura mater, 5 galea, 6 cranialized frontal sinus

• Pericranial flap

To provide a secure occlusion of any skull base defect, a vascularized, pedicled pericranial flap is swung over the osseous border of the frontofacial compartment, spread out over the reconstructed floor of the anterior cranial fossa over an area as extensive as possible and fixed with sutures.

If necessary, a supplementary pedicled temporal fascia-flap can be inserted uni- or bilaterally approaching from lateral (Jackson et al. 1982, 1986; Kessler 1983; Price et al. 1988; Weerda 1995; Imhof 2000).

In the subsequent reconstruction of the frontal bone, an inferior, transverse bone-slit is left to avoid any compression to the wrapped-over, vascularized pericranial flap (Figs. 9.5 and 9.6).

• Bone grafts

Under certain circumstances there is an indication for a simultaneous closure of osseous skull base defects with autogenous cancellous or cortico-cancellous bone grafts (Probst and Tomaschett 1990).

- Increased liquorrhea from a CSF fistula (e.g., oncoming cerebral atrophy, opening of the basal cisternae and the ventricular system).
- Unfavorable fistula localization (in the region of a low positioned cribriform plate)
- In very wide bony fracture gaps, an additional autogenous bone graft is integrated to bridge the defect; the region is subsequently covered with an autogenous muscle patch. Particularly in the case of large osseous skull base defects with the risk of necrosis of the above-lying duraplasty (e.g., after extensive ethmoidectomy) a stabilizing layer of autogenous bone should be inserted to avoid herniation of the orbital gyri (Samii and Draf 1989; Stoll 1993; Frodel 2002).

Either calvarial bone from the inner table of the skull or cancellous bone may be used as bone grafts (Stanley and Schwartz 1989) (Figs. 9.7-9.9).

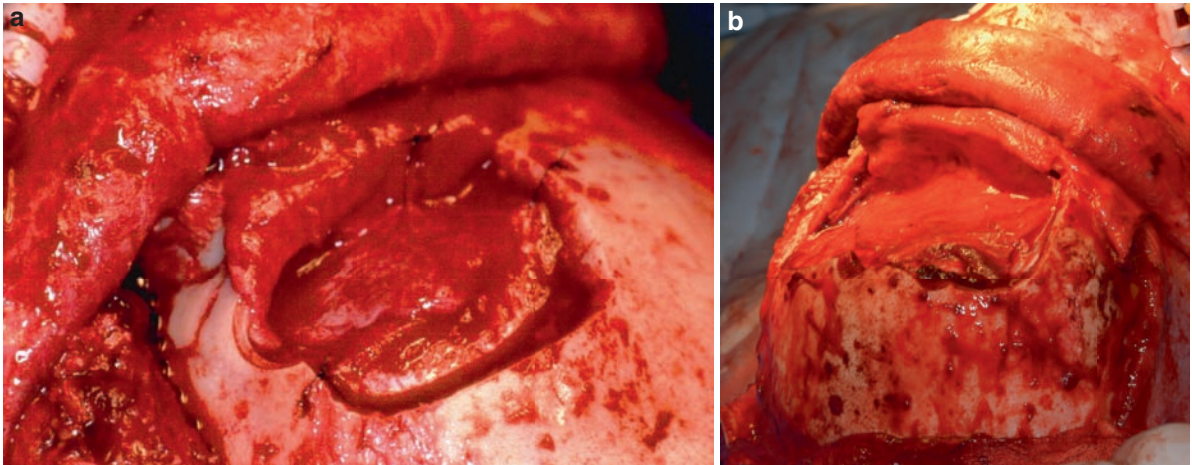


Fig. 9.6 Extradural treatment of fractures of the frontal skull base: insertion of pericranium flaps to cover the frontobasal area (a) and the frontal dura (b)

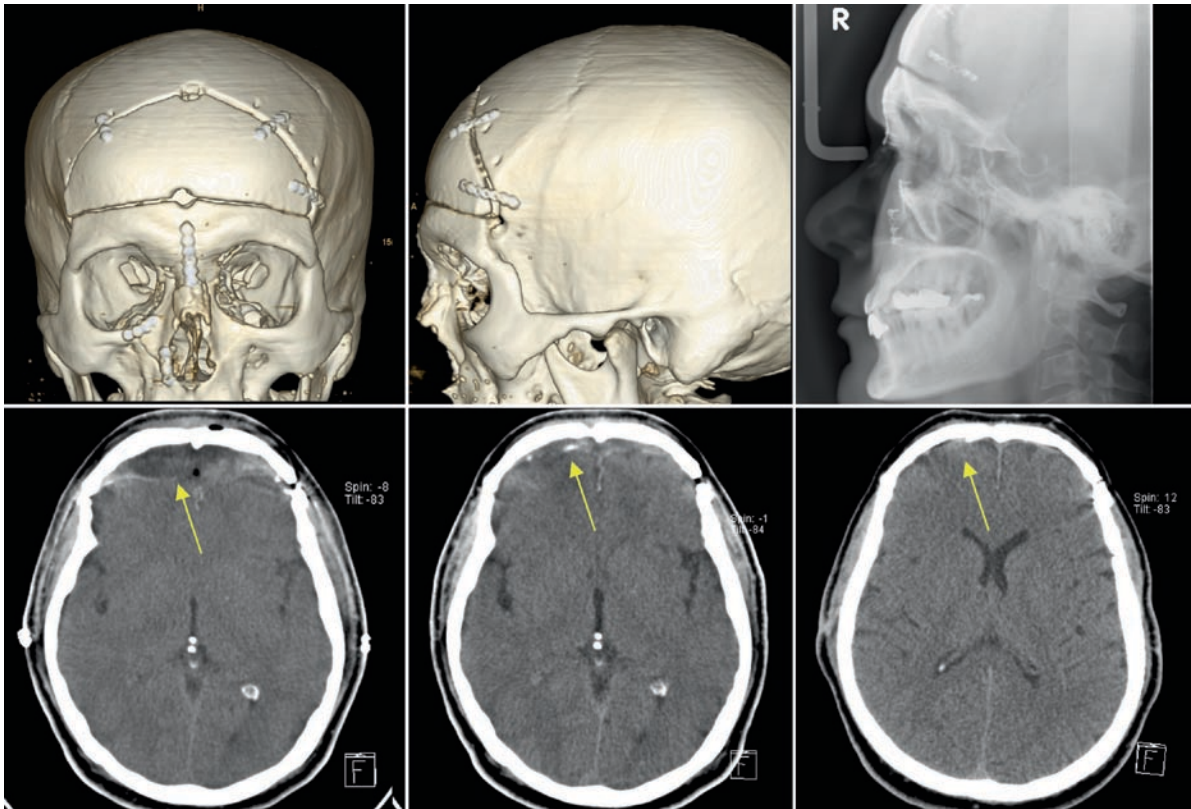


Fig. 9.8 Cranialization of the frontal sinus after fracture of the midface with frontal skull base fragmentation. The posterior wall of the frontal sinus has been removed. The brain slowly

expands and partially fills the additional space. The residual space is filled with fat and scar tissue (arrow). Calcifications at the level of the dura

Fig. 9.7 Schematic diagram showing expansion of the brain after cranialization of the frontal sinus and reconstruction of the frontal skull base (mod. a. Prein et al 1998). (a) The frontal skull base is reconstructed with cancellous bone or split skull grafts, which are covered with a pericranial flap (galea frontalis flap) in order to securely seal the intracranial cavity from the nose. (b) The brain slowly expands and fills the additional space

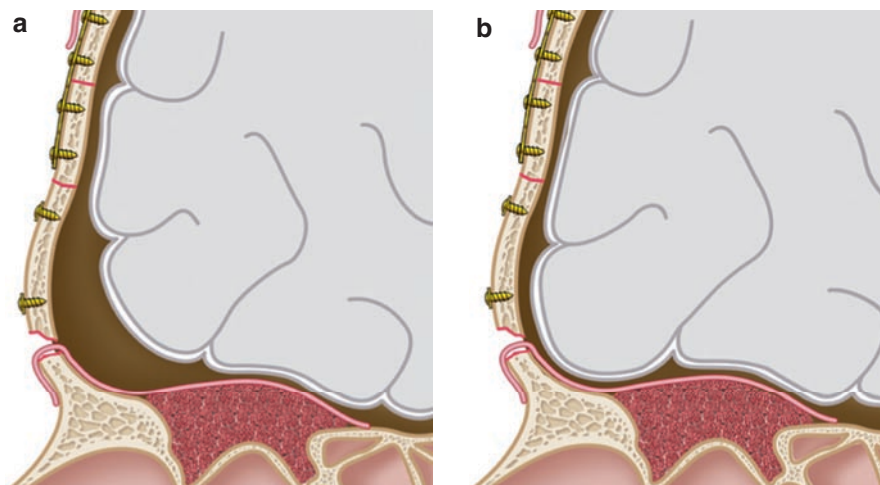
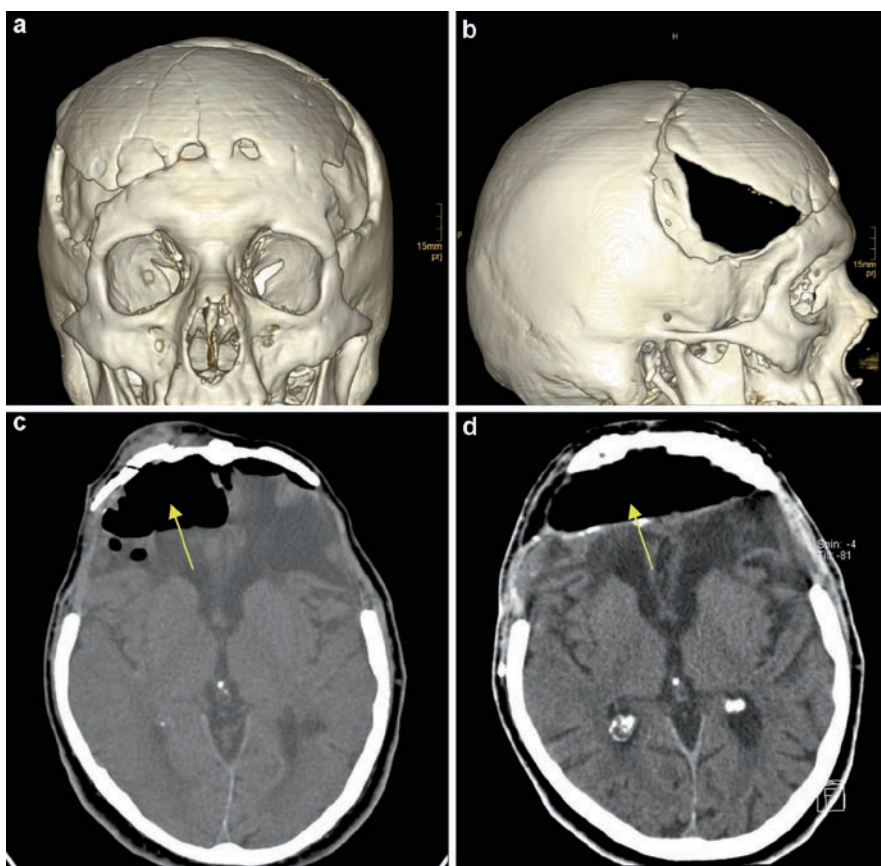


Fig. 9.9 (a, b) Repeated frontal trauma. There are large bilateral frontotemporal bone defects after previous trauma, leaving a shield-like frontal bone with limited resistance to trauma. (c) Frontal bone fracture. Dural injury with pneumocephalus. Pre-existing posttraumatic brain defects in both frontal lobes. (d) Postoperative result after closure of the dura. Persisting extradural pneumocephalus. The frontal lobes do not expand sufficiently because of the posttraumatic brain defects



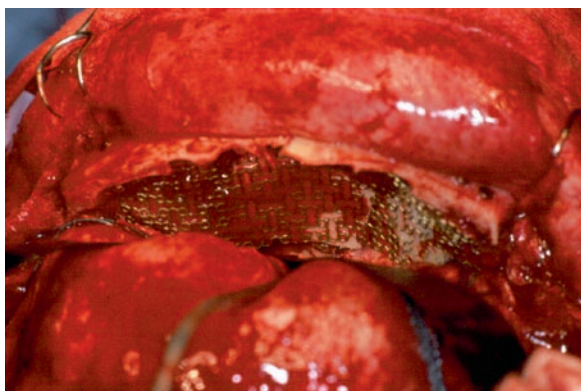


Fig. 9.10 Reconstruction of the frontal skull base with autogenous bone transplants in combination with a contouring and stabilizing titanium mesh

If several bone grafts are required to reconstruct the skull base in extensive defects (e.g., following gun shot injuries), an extradurally applied microtitanium mesh may be used to stabilize the grafts and provide the contour of the skull base (Hardt et al. 1994; Reinert and Gellrich 1997; Deinsberger et al. 1998; Mick 1999; Kutenberger and Hardt 2001).

The microtitanium mesh is cut and designed according to the defect form and modelled onto the frontal skull base. The mesh is then fixed with miniscrews lateral to the orbital roof in order to stabilize the bone fragments or grafts, which have been inserted basally between the mesh and the skull base. The titanium mesh itself is totally covered with a wide pericranium flap, so that the entire skull base is covered right up to the edge of the wing of the sphenoid bone (Mick 1999) (Fig. 9.10).

9.3.3.2 Intradural Skull Base Occlusion

The dura defects may not be extended or the borders of the basal dura injuries be exposed until the fronto-basal, frontofacial, and zygomatico-orbital structures have been completely repositioned and stabilized. Subsequently, following intradural exposure of the boundaries, the dural leakage is occluded using a pericranium/muscle-patch. In case of severe dural injuries, a pedicled pericranial flap is then applied overlapping the basal dural injury.

The exposed frontal sinus is simultaneously covered by the superiorly placed pericranial flap. The pericranial flap is fixed to the basal dura using interrupted

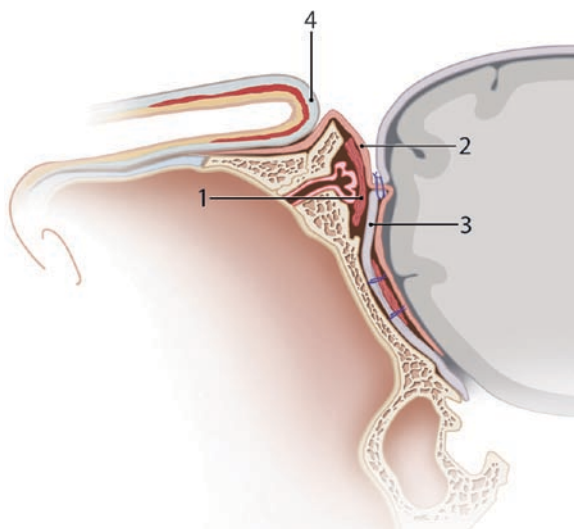


Fig. 9.11 Schematic diagram illustrating intradural treatment of anterior skull base fractures. The pericranial flap is placed intradurally over the basal dura after obliteration of the nasofrontal duct with a muscle patch (mod. a. Imhof 2000). 1 Muscle patch covering the invaginated nasofrontal duct, 2 pericranial flap, 3 dura mater, 4 galea

sutures along the dural incision borders and circularly around the frontobasal defect. The frontal lobe of the brain is relocated to the anterior skull base now covered with the pericranial flap.

The remaining dura defect will be closed by suturing the pericranial flap to the superior-cranial dural border (Ewers et al. 1995; Imhof 2000) (Fig. 9.11).

If indicated an ICP probe is inserted in the subdural space, ideally through a separate high frontal parasagittal burr hole (Imhof 2000).

The state of the paranasal sinuses should be controlled 4-8 weeks postoperatively following treatment of a frontal skull base injury. In case of congestion or insufficient drainage of the ethmoid cells, an endonasal revision may follow electively as a second procedure (Wigand 1989; Ernst et al. 2004).

9.4 Skull Base Treatment/Own Statistics

In skull base reconstruction, several layers are frequently placed over each other, so applying different techniques in one patient.

In our own patients, skull base treatment resulted in 68% having a combined *dural suture and duraplasty*

and in 87% with an additional *pericranial flap*. Muscle patches from the temporalis muscle were used in 72% to occlude the nasofrontal duct as well as covering the debrided ethmoid cells.

Larger bony defects in the basal region were occluded with *autogenous bone fragments or calvarial grafts* in 37%. In 61% of our patients, the comminuted dorsal wall of the frontal sinus was resected and a *cranialization* of the frontal sinus was carried out (Neidhardt 2002).

Techniques of skull base-dura reconstruction in our own craniofacial fractures (Neidhardt 2002)

Dural suture and duraplasty	68%
Pericranial flap	87%
Muscle patches	72%
Bone grafts	37%

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Reconstruction of bony defects with autogenous bone grafts is an integral part of modern treatment concepts in traumatology of the craniofacial skeleton (Tessier 1982; Jackson et al. 1983, 1987; Gruss et al. 1985; Gruss 1986; Marentette 1988; Ilankovan and Jackson 1992; Hammer 1995, 2002; Zins et al. 1995, 1999; Prein 1998; Maniglia et al. 1999; Dempf et al. 1998; Donald 1998; Frodel 2002; Greenberg and Prein 2002).

Whereas accurate primary bone defect coverage can achieve excellent aesthetic and functional results, secondary reconstruction often does not provide satisfactory results. The indication for autogenous bone grafts should, therefore, be liberally made during the primary intervention (Gruss 1986; Jackson et al. 1987; Salyer 1989; Whitaker 1989; Ilankovan and Jackson 1992; Manson 1994, 1998a; Hammer 1995; Dempf et al. 1998; Frodel 2002).

10.1 Indications

There are several indications for using bone grafts to reconstruct osseous defects:

10.1.1 Midface

(Manson et al. 1985; Marentette 1988; Gruss and Mc Kinnon 1986; Serletti and Manson 1992; Frodel 2002)

- The primary and simultaneous insertion of bone grafts to cover *bony defects* or to *bridge gaps in the area of the load-bearing maxillary pillars* is a radical improvement in maxillofacial fracture treatment.

In cases of substantial bone loss, the integration of bone grafts results in an improved *fracture stabilization* and has a preventative function against midfacial collapse (midfacial elongation or shortening) (Klotch and Gilliland 1987).

- On the other hand, disfiguring facial contours from scarring or soft tissue contractions overlying the deficient osseous structures can also be avoided (Gruss et al. 1985; Manson et al. 1985; Gruss and Mc Kinnon 1986; Gruss and Philipps 1989; Mathog 1992; Salyer 1992; Härle et al. 1999; Frodel 2002).

Due to excellent stability achieved by plate and screw fixation, the necessity of primary bone grafting has been reduced (Stanley and Schwartz 1989; Härle et al. 1999).

10.1.2 Frontofacial Region (Gruss and Mc Kinnon 1986; Serletti and Manson 1992; Frodel 2002)

- Untreated fronto-cranial bone defects do not heal spontaneously and result in soft tissue retractions and conspicuous deformations, especially in aesthetically important regions.
- A complete osseous reconstruction *protects the brain and provides a functional platform for the mimical muscles*.
- Apart from anatomically correct repositioning and stabilizing of all fragments, it is necessary to fill remaining gaps, defects, and trepanation holes to avoid subsequent contour irregularities (Jackson et al. 1986; Sailer and Graetz 1991; Dufresne et al. 1992; Hardt et al. 1992, 1994, Lee et al. 1998).

10.1.3 Orbital Region (Jackson et al. 1986; Serletti and Manson 1992; Hammer and Prein 1998)

- Frontal defects in the eyebrow region interfere with *eyelid mobility and functionally disturb perspiration*. An efficiently reconstructed supraorbital arch protects the globe.
- In *orbital roof defects*, bridging the gaps with convex bone transplants prevents the development of an enophthalmus and the transmission of cerebral pulsations to the orbital soft tissues and a potential cerebral prolapse (Mohr et al. 1994).
- Orbital wall defects have to be reconstructed with bone grafts, mesh systems or alloplastic sheets to avoid herniation of orbital soft tissue with subsequent enophthalmus or muscle imbalance or incarceration (Korneef 1982; Gruss et al. 1985; Ilankovan and Jackson 1992; Mathog 1992; Serletti and Manson 1992; Sugar et al. 1992; Merten and Luhr 1994; Piotowski and Beck-Manntagetta 1995; Gehrke et al. 1996; Hammer and Prein 1998; Dietz et al. 2001; Dempf et al. 2001).

Prolapsed periorbital soft tissues are carefully relocated and incarcerated eye muscles are released and replaced. Bone grafts may both be directly integrated and wedged into the defect in a self-stabilizing manner or as a cantiever, stabilized by a microplate at the orbital margin (Gruss et al. 1985; Antonyshyn et al. 1989; Mathog 1992; Hammer 1995; Reinert and Gellrich 1997; Markowitz and Manson 1998; Manson 1998b; Hammer 2002; Greenberg and Prein 2002; Frodel 2002).

10.2 Autogenous Bone Grafts

Due to the proximity to the operation field in a coronal approach, it is advisable to preferentially harvest autogenous bone grafts from the calvarium (Jackson et al. 1982; Dufresne et al. 1992). Various bone grafting techniques are used to bridge bony defects.

10.2.1 Split Calvarial Grafts

These grafts are obtained by splitting bicortical calvarial bone.

The *inner table* is used as a graft to reconstruct defects and can in itself be variably altered in size and shape, whereas the *outer table* is used to restore the skull contour of the donor side. Split calvarial grafts are particularly used in large defects (Froberg and Deatherage 1991; Dufresne et al. 1992; Salyer 1992; Hardt et al. 1994; Greenberg and Prein 2002; Frodel et al. 1993; Frodel 2002) (Fig. 10.1).

Technique of harvesting split calvarial grafts (Kellman and Marentete 1995)

Placing craniotomy holes and connecting osteotomies
Detaching the dura and extracting the bicortical bone flap
Splitting the bicortical bone flap through the diploic space into inner and outer table using microsaw and chisels

• Inner table grafts

Tabula interna grafts have the advantage of providing a sufficient amount of donor bone without leaving a cosmetic defect in the calvarium. Inner table grafts are most commonly harvested from the inner table of the skull when intracranial-extracranial procedures are being performed.

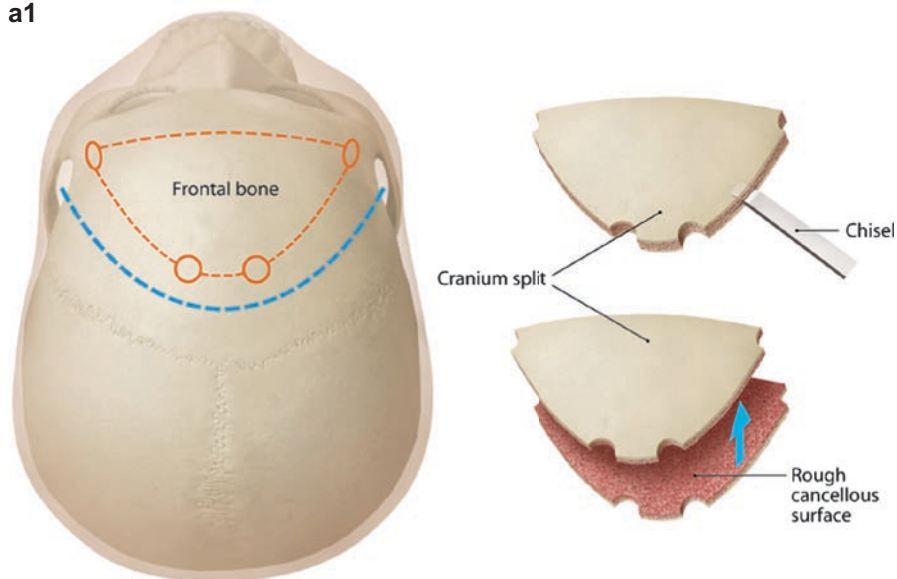
According to Kellman and Marentete (1995) a piece of skull bone analogical to the size of the defect has to be determined and harvested to reconstruct the defect. For resecting the inner table of the double-layered skull bone, an oscillating or reciprocating saw may be used. After outlining the size of the graft on the inner table, the diploic layer is divided carefully with the saw. After circular incision, a chisel may be used to separate inner and outer table (Figs. 10.2 and 10.3).

• Outer table grafts

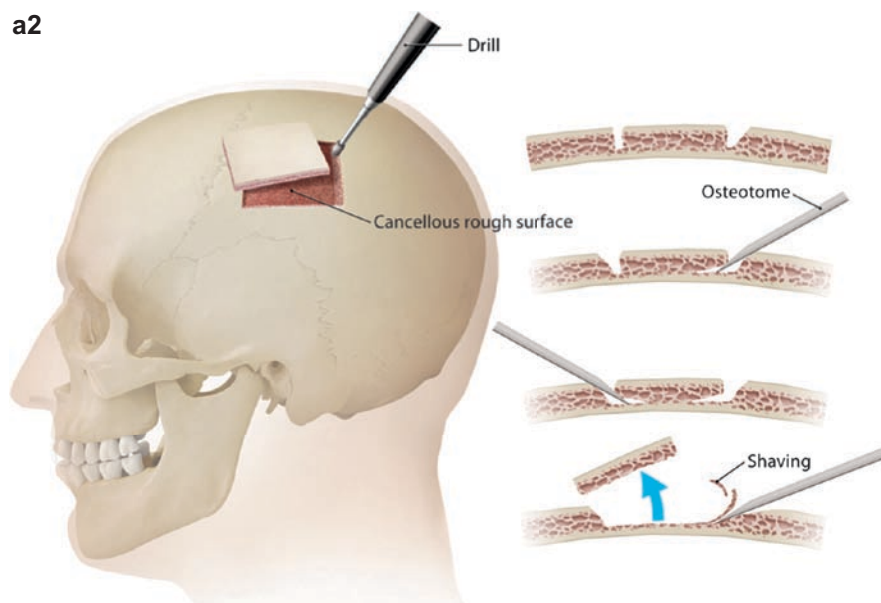
Grafts of the outer table can be harvested without performing a craniotomy.

The desired graft size is outlined with a burr until the diploic space is reached. Then the marked bone graft is removed with special chisels (sledge-shaped osteotomes), an oscillating saw or a piezo surgical device. The parietal skull region — as the preferred donor site — is generally sufficiently thick to harvest outer

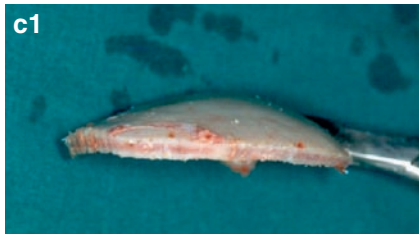
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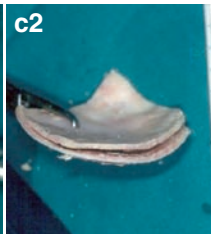
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c1



c2



c3

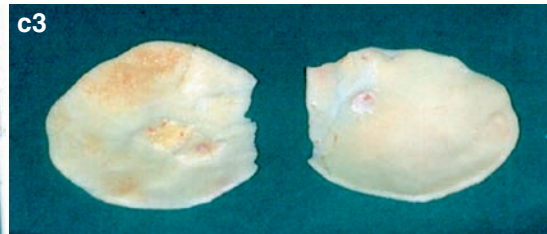


Fig. 10.1 Technique of harvesting calvarial bone grafts (mod. a. Kellman and Marentete 1995). (a) Splitting the fronto-temporal bone flap with chisels or saw to obtain monocortical calvarial grafts. (b) Harvesting of monocortical bone grafts from the outer table of the skull. After creating a groove with a round burr, the split skull graft is elevated with chisels or a sagittal saw. The

separation requires a precise technique with correct insertion of the osteotome into the diploic space at a correct angle, parallel to the inner and outer table. (c) Splitting the bicortical calvaria in the interconnecting diploe into monocortical tabula externa and tabula interna grafts

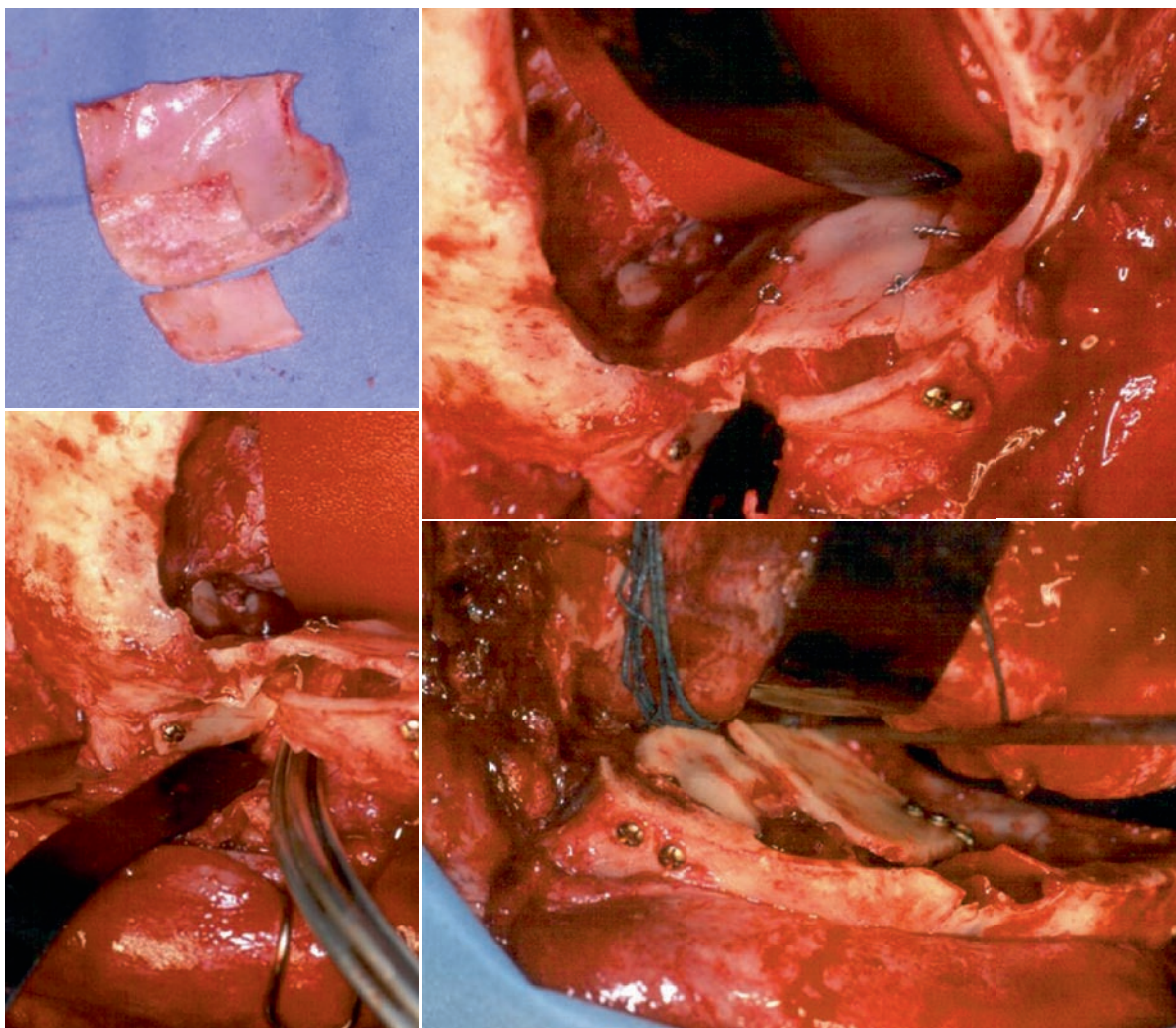


Fig. 10.2 Reconstruction of an extensive orbital roof-defect with a split calvarial graft from the inner table of the skull. The bone graft is fixed with wire osteosynthesis or alternative with microplates

table grafts. The defect can be filled with bone cement if necessary (Jackson 1986; Jackson et al. 1987; Froberg and Deatherage 1991; Hardt et al. 1992; Dempf et al. 1998).

The *donor site* should be located on the parietal bone between the temporal line and a point 1.5 cm lateral to the sagittal suture. The bone below the temporal line is too thin to allow safe harvesting of the graft. It is crucial to avoid the midline, because then the superior sagittal sinus is located beneath the sagittal suture and inadvertent penetration of the inner table during graft harvest could result in laceration of the sinus, which would result in significant

bleeding and possibly an air embolism and/or sinus thrombosis.

Donor site bleeding may be controlled by using either bone wax or a resorbable collagen sponge. During harvesting, small emissary veins may be encountered in the diploic layer, causing significant bleeding. If this occurs early during the harvesting of the graft, then bone wax is placed at the edges of the graft to control the bleeding, and another donor site may be chosen.

However, if an emissary vein is encountered during harvesting the outer table, then the procedure should be finished as quickly as possible and the graft

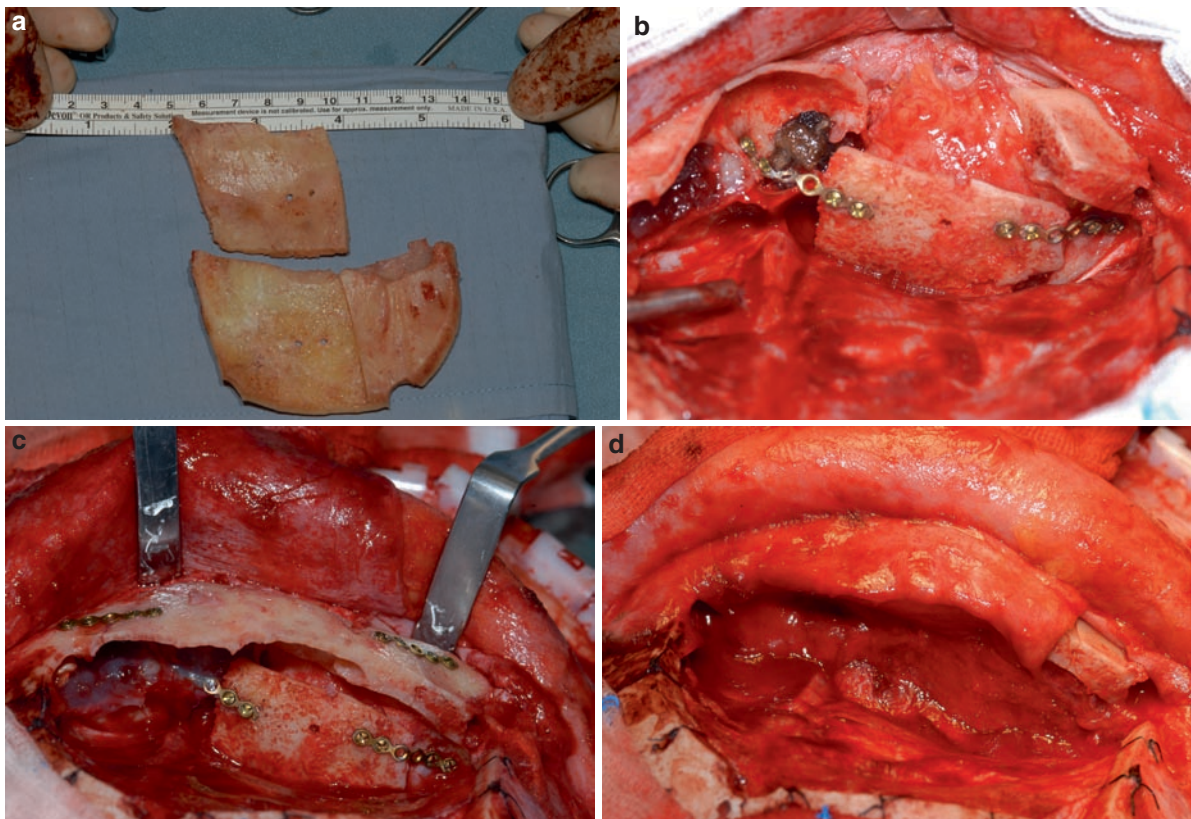


Fig. 10.3 Reconstruction of an extensive defect of the orbital roof with split calvarial graft (*inner table*). (a) Harvested graft. (b) Reconstructed orbital roof after frontofacial osteotomy, cranial debridement and canalization of the frontal sinus. The bone

graft is fixed with two miniplates. (c) Reintegration of the fronto-facial bone segment. (d) The reconstructed orbital roof is covered with a pericranial flap

removed. Immediate hemostasis is accomplished by using bone wax.

Technique of harvesting monocortical outer table transplants (Kellman and Marentete 1995)

The maximum circumference of the transplant is outlined with a large round burr until the diploic space is reached

Elevation of the graft is carried out using a flexible saw, chisels, or piezosurgical device

The convex form of the thin outer table of the skull facilitates fitting of the graft particularly into regions of disrupted concave midfacial contours (orbital wall defects). Designed bone grafts can be used to reconstruct the load-bearing facial pillars or the nasal dorsum.

Grafts harvested in an anteroposterior direction are straighter and best suited for the reconstruction of the medial buttresses and nasal dorsum. Grafts oriented in a superoinferior direction along the lateral skull are more curved and generally better suited for reconstruction of the lateral buttresses in the zygomatic region and the orbital walls.

The carefully inserted bone grafts have to be stabilized with mini- or microplates. The local morbidity rate, mostly in the form of small wound complications, may reach 4.6% (Ilankovan and Jackson 1992) (Fig. 10.4).

• Graft resorption

In contrast to iliac crest or costal grafts, the *membranous, desmal* calvarian bone grafts exhibit a considerably lower resorption rate (Withacker 1989). The *enchondral* iliac and costal grafts display resorption rates of 60-80% of their original volume, whilst the desmal calvarian

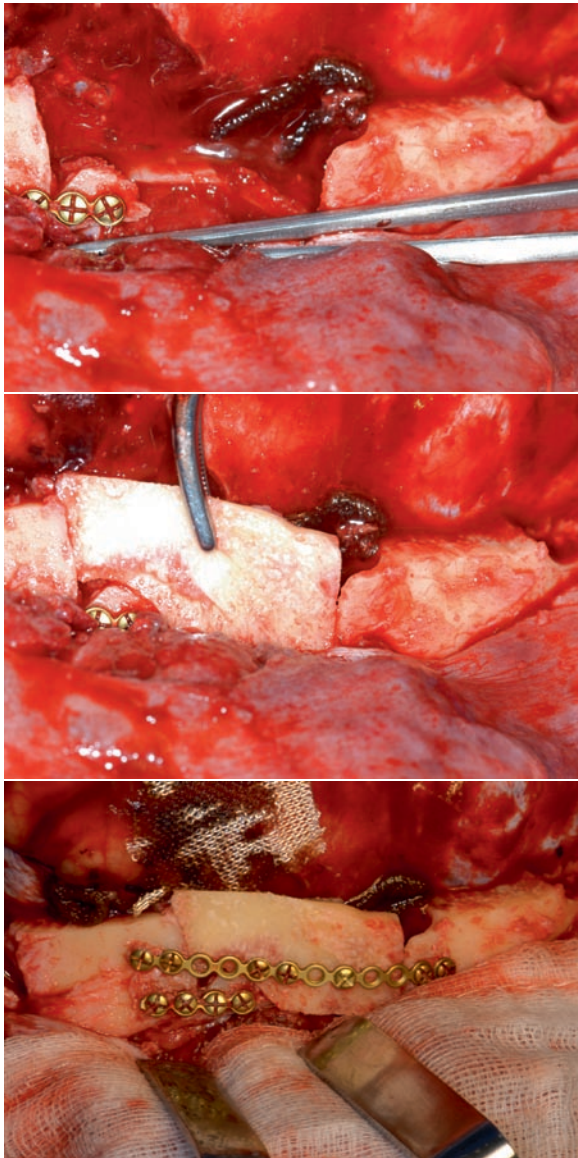


Fig. 10.4 Reconstruction of the supraorbital rim with a split calvarial graft from the outer table of the skull

bone grafts merely lose 17–19% of their volume (Smith and Abramson 1983; Zins and Withaker 1983; Zins et al. 1984; Craft and Sargent 1989).

The minimal resorption of calvarial bone grafts offers a superior form consistency and subsequently more reliable aesthetic results. Consequently, as a result of the difficulty in estimating resorption in the graft area, in the majority of cases iliac crest and costal bone grafts have been abandoned (Fowler et al. 1995) (Fig. 10.5).

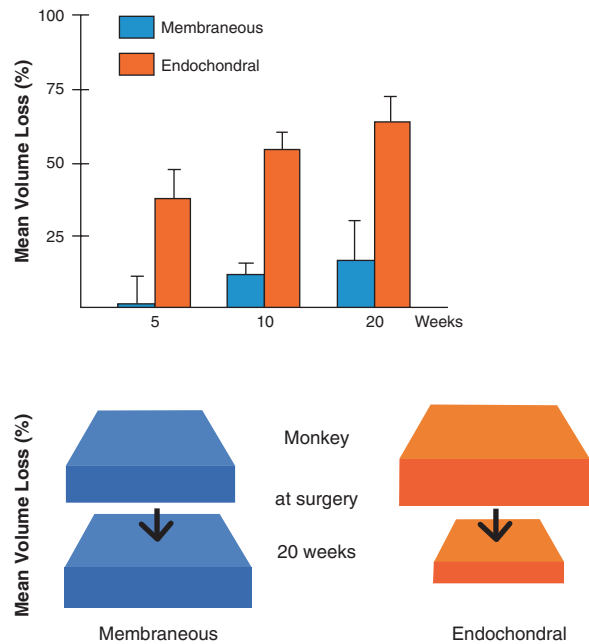


Fig. 10.5 Volumetric comparison of resorption between endochondral and membranous bone grafts (mod. a. Zins and Withaker 1983)

The degree of graft resorption is also influenced by the stability of the bone graft. A rigid fixation with miniplates noticeably reduces the extent of resorption (Philipps and Rahn 1988). A rigidly fixed membranous bone graft experimentally maintains a 10% greater volume than a nonfixed membranous bone graft. Rigidly fixed endochondral bone grafts maintain 75% of their volume in comparison with 15% in nonstabilized endochondral grafts (Philipps and Rahn 1990).

Despite the experimentally low to moderate graft resorption in desmal bone grafts with adequate osteosynthesis, clinically there may be an unexpectedly greater resorption in the long-run (Fowler et al. 1995).

10.2.2 Bone Dust/Bone Chips

There is experience in filling defects with bone dust (Jackson et al. 1986; Dufresne et al. 1992; Hardt et al. 1994). Small residual defects such as craniotomy holes can be filled with bone dust collected intraoperatively. Resorbable membranes or titanium craniotomy rosettes can also be used to achieve stabile contours (Greenberg and Prein 2002).

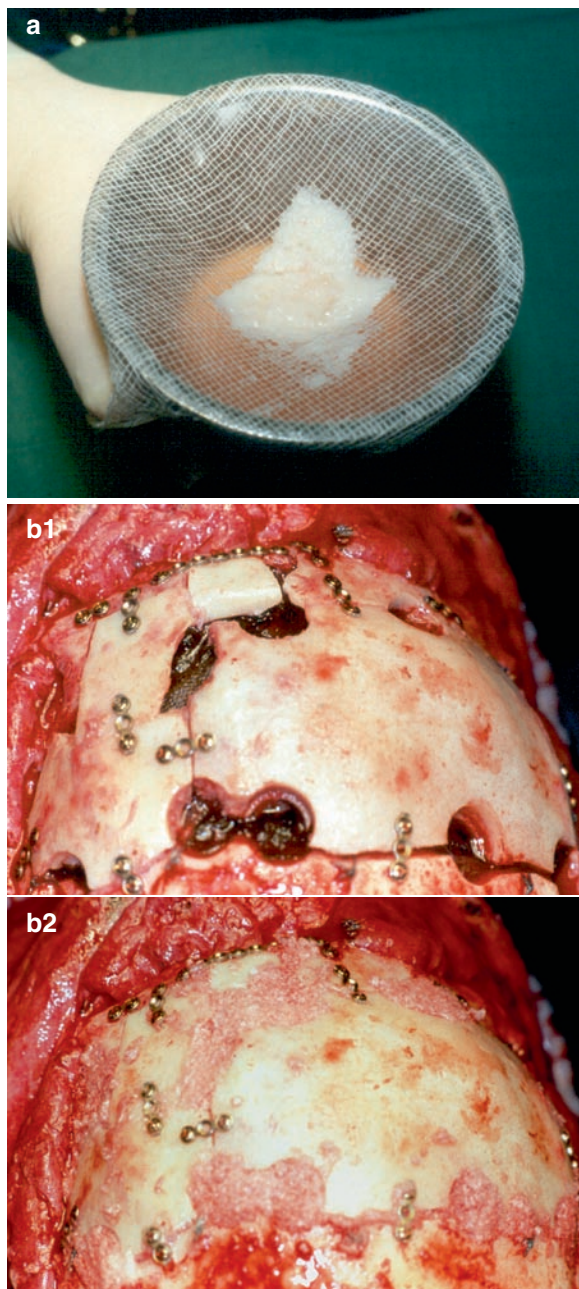


Fig. 10.6 Filling cranial bone defects with bone dust. (a) Bone dust (bone particles) collected during drilling of craniotomy holes. (b) After reinsertion of the frontal bone flap, burr holes and small bone defects are filled with bone dust

- Bone dust

The majority of residual defects filled with bone dust exhibit distinct resorption. The bone is replaced by connective tissue, often with irregular to concave depressions, resulting in an uneven skull surface.

An incomplete spontaneous osseous bridging is clinically and histologically found only at the level of the inner table (Hardt et al. 1994).

There are comparable results when using bone chips, which are attained from crushing tiny, nonintegrable bony fragments in a bone-mill (Hardt et al. 1994) (Fig. 10.6).

- Bone dust and membrane coverage

A few months after covering bone dust-filled defects with reabsorbable membranes (GORETEX), a smooth, coherent, niveau of regenerative connective tissue is formed under the membrane. Again, one only finds an incomplete, defect-bridging osseous regeneration at the level of the inner table. The regenerative capacity of bone is age-dependant (Hardt et al. 1994) (Figs. 10.7 and 10.8).

- Bone dust and titanium rosettes

The combination of bone dust and craniotomy rosettes produces clinically equivalent results. After removing the titanium rosettes, there is connective tissue with or without minimal niveau loss. The mostly incomplete osseous regeneration is exclusively limited to the level of the inner table (Hardt et al. 1994) (Fig. 10.9).

10.2.3 Autogenous Grafts from the Iliac Crest

Cortico-cancellous grafts from the iliac crest are rarely used to reconstruct skull bone defects. However, the iliac crest is an excellent reservoir for large amounts of cancellous bone, which may be needed for frontal sinus obliteration or frontobasal reconstruction.

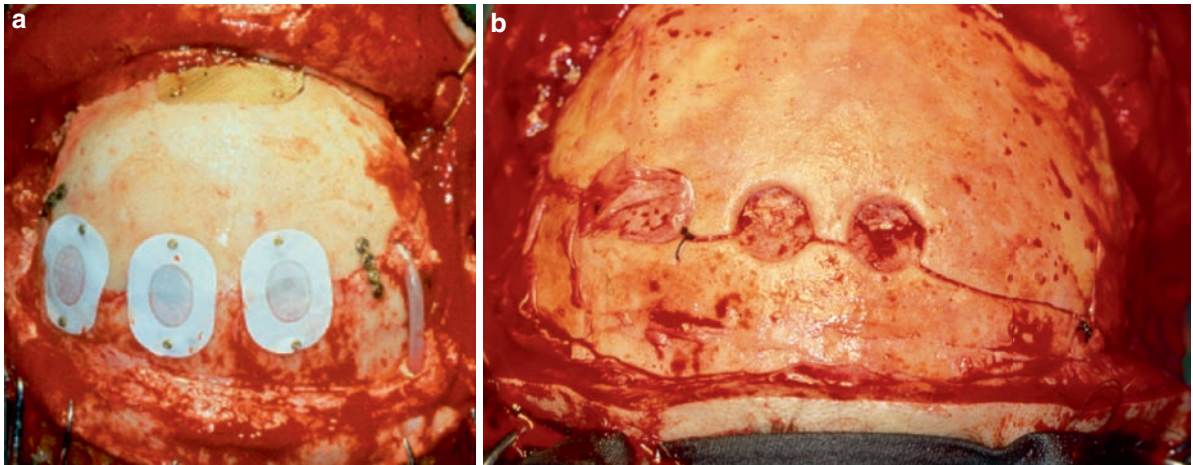


Fig. 10.7 (a) Burr holes filled with bone dust and covered with GORETEX membranes, which are fixed with miniscrews. (b) Integrated GORETEX membranes are removed 12 months postoperatively. The burr holes are closed with bone at the level of the inner table

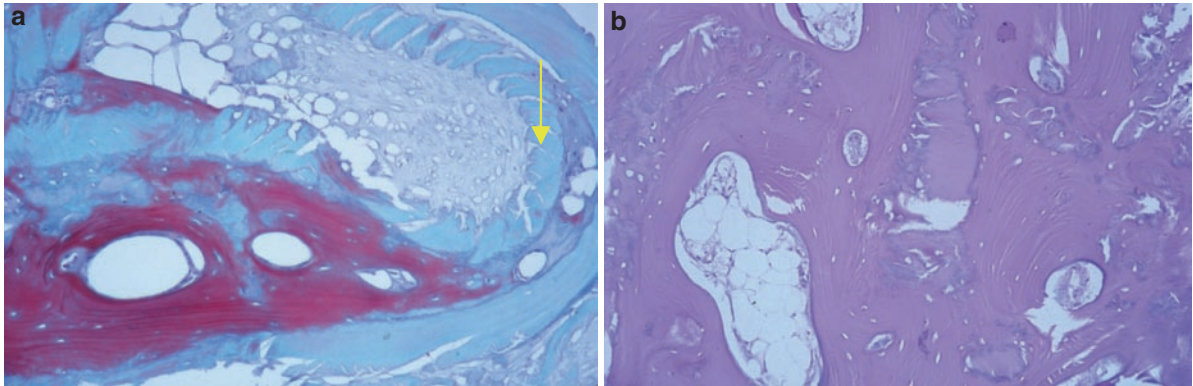


Fig. 10.8 Histological section through a burr hole which was filled with bone dust and covered with a GORETEX membrane. (a) Newly-formed bone with remnants of the membrane (*arrow*)

(160 $\times$). (b) Almost complete reconstruction of a trepanation defect filled with vital bone (100 $\times$)

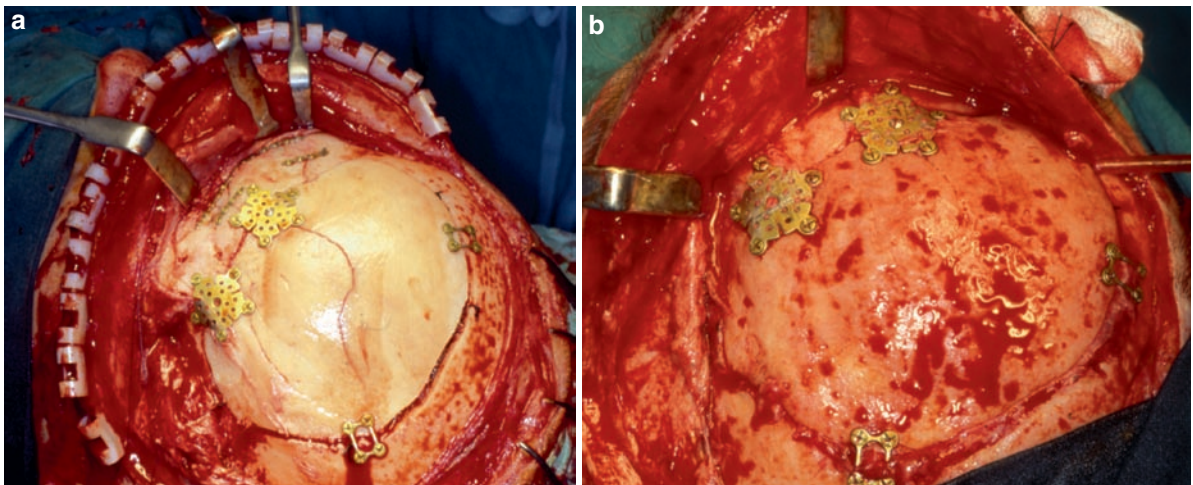


Fig. 10.9 Covering burr holes with titanium rosettes. (a) Intraoperative view after insertion of the titanium rosettes. (b) Twelve-month follow-up with properly integrated rosettes

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11.1 Biomechanics: Facial Skeleton

The craniofacial skeleton comprises 22 different bones and exhibits remarkable stability, although it is partly composed of pneumatized bones. The bony areas delimit the cranial vault, the orbital cavities, the paranasal sinuses and the oral cavity. Within the craniofacial skeleton there are thicker load-bearing and thinner nonload-bearing bony regions (Ewers et al. 1995).

The osseous facial skeleton is supported by three *vertical struts*:

- Naso-maxillary-frontonasal strut
- Zygomatico-maxillary strut
- Pterygo-maxillary strut

and three *horizontal transverse struts*:

- Maxillary alveolar process
- Infraorbital-nasal rim
- The fronto-cranial skull base/frontofacial bandeau

Their arrangement corresponds to that of the micro-trajectory configuration of cancellous bone (Schilli et al. 1981; Manson et al. 1980; Manson et al. 1990; Härle et al. 1999; Ernst et al. 2004) (see Figs. 1.10 and 1.11).

- As a bony framework, the transverse and vertical struts determine the vertical facial height, the transverse facial width, the sagittal midfacial position and, consequently, the symmetry and projection of the facial skeleton (Manson 1986; Härle et al. 1999).

Fractures with dislocation of the midfacial complex in the sagittal, transverse, and vertical dimensions induce the loss of the three-dimensional midface projection.

If these structures are not adequately repositioned and stabilized, this consequently results not only in varying degrees of *disfiguration and deformation* but also in *functional disability and a relevant danger of*

infection (Gruss et al. 1989; Joss et al. 2001) (Figs. 11.1 and 11.2).

Consequences of defective positioning of these skeletal structures are:

- Occlusal dysfunction — dysgnathic maxillary position
- Dish face - midfacial retrusion — pseudoprognathia
- Occlusal disturbances — open-bite
- Elongated or shortened midface
- Broadening of the facial skeleton

Functional impairments

- Obstruction of the nasal airways with reduced aeration of the paranasal sinus system
- Insufficient function of the naso-lacrimal duct
- Ophthalmic problems — diplopia, enophthalmus, etc.
- Masticatory insufficiency
- Chronic pain
- The anatomical reconstruction and stabilization of the facial struts is essential in reestablishing the normal midfacial relation with the skull base and for restoring the midfacial projection, including normal occlusion (Rowe and Williams 1985; Gruss et al. 1985a, b; Gruss and Mc Kinnon 1986; Klotch and Gilliland 1987; Gruss and Philipps 1989; Manson et al. 1995; Weerda 1995; Joss et al. 1996, 2001; Manson 1998)

11.2 Principles of Biomechanical Reconstruction

An understanding of the structure and biomechanics of the maxilla and midface, accompanied by an anatomically orientated therapy, has resulted in significant

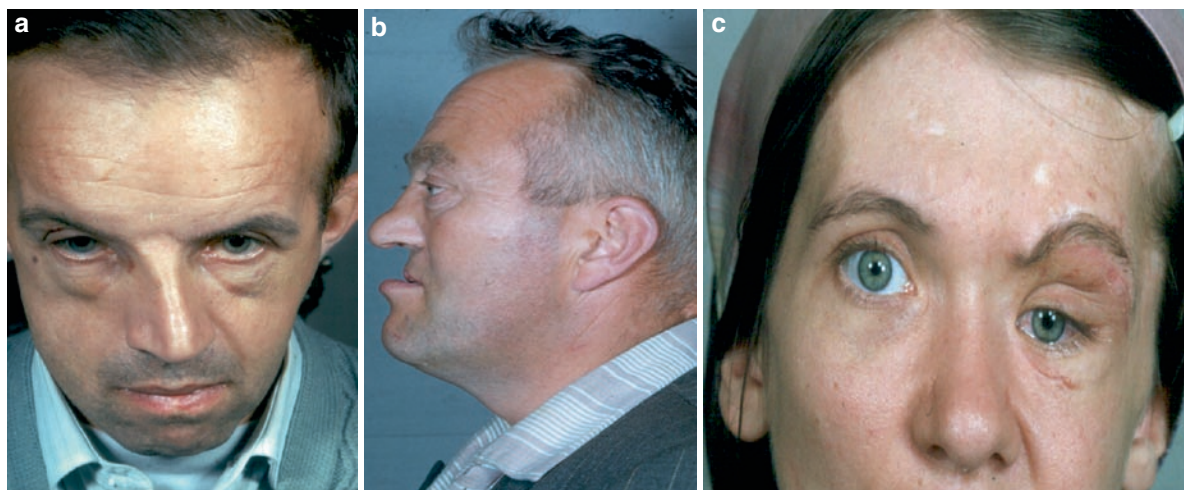


Fig.11.1 Malalignment of midface fractures with loss of facial symmetry. (a) Depression of right zygomatic region, deviation of osseous nasal pyramid and enophthalmus following zygomatico-orbital and naso-maxillary fracture. (b) Dish-face deformity following a severe central midface fracture (Le Fort I and II).

(c) Severe asymmetry of the lateral midface with malalignment of the bony orbit, loss of orbital soft tissue with low-lying globe, enophthalmus and significant disturbance of eye motility after comminuted zygomatico-orbital fracture on the left

Fig.11.2 Facial asymmetry after severe cranio-orbito-nasal fracture with comminution of the left orbital walls. Alteration in globe position (enophthalmus), disturbance of eye motility (diplopia), narrow palpebral fissure, pseudoptosis, deviation of nasal skeleton, increase of intercanthal distance



improvements in maxillofacial traumatology (Manson et al. 1980, 1985; Manson 1986; Gruss 1990; Dufresne et al. 1992; Prein et al. 1998; Hausamen and Schierle 2000; Ward-Booth et al. 2003).

The midfacial framework composed of an external and internal skeletal frame is the key to rigid fixation of the midface skeleton using varying plate thicknesses. A primarily correct and stable reconstruction of the osseous structures exhibits numerous advantages:

- Aesthetic improvement and functional stability
- Reduced risk of infection
- Uncomplicated fracture healing
- Transplant healing with minimal resorption
- Reduction of pain

11.2.1 External Midfacial Skeletal Framework

Gruss and Mc Kinnon (1986) stress the importance of a precise initial reconstruction of the external skeletal frame in order to establish correct facial dimensions. This midfacial framework comprises the transverse frontofacial junction, the zygomatico-orbital complex and the external, lateral midfacial strut (lateral zygomatico-maxillary strut).

- The frontofacial and zygomatico-orbital regions biomechanically form an important subcranial structure at the intersection between the viscer- and

neurocranium and are, therefore, important corner struts for the three-dimensional reconstruction of the subordinate midface complex

The *frontofacial* region plays a key role in a correct transverse and sagittal reconstruction of facial width and depth. Dislocated fractures of this compartment result in a loss of anatomical orientation for reconstructing the fractured midfacial complex (Sailer and Graetz 1991) (Fig. 11.3).

The *zygomatiko-facial compartment* forms the basis for reconstructing facial width and orbital depth and the *fronto-facial compartment* for the sagittal position of the naso-orbito-ethmoidal (NOE) complex (Prein et al. 1998). Only if the fronto-facial junction is anatomically correctly reconstructed, is it possible to correctly position the zygomatico-maxillary and naso-ethmoidal complexes and to reconstruct the orbital cavity (Wolfe and Berkowitz 1989; Gruss et al. 1990; Kraft et al. 1991; Hardt et al. 1992; Prein et al. 1998).

The *zygomatiko-orbital complex* influences facial width as well as sagittal projection of the midface (Gruss et al. 1992; Gruss 1995; Manson et al. 1999; Brisett and Hilger 2005). Postero-lateral displacement of the zygomatico-orbito-maxillary complex results in broadening of the midface with a postero-lateral curvature of the zygomatic arch region and reduction of facial antero-posterior projection (Brisett and Hilger 2005). A correct sagittal midfacial projection is assured if reconstruction of the zygomatic arch is anatomically correct.

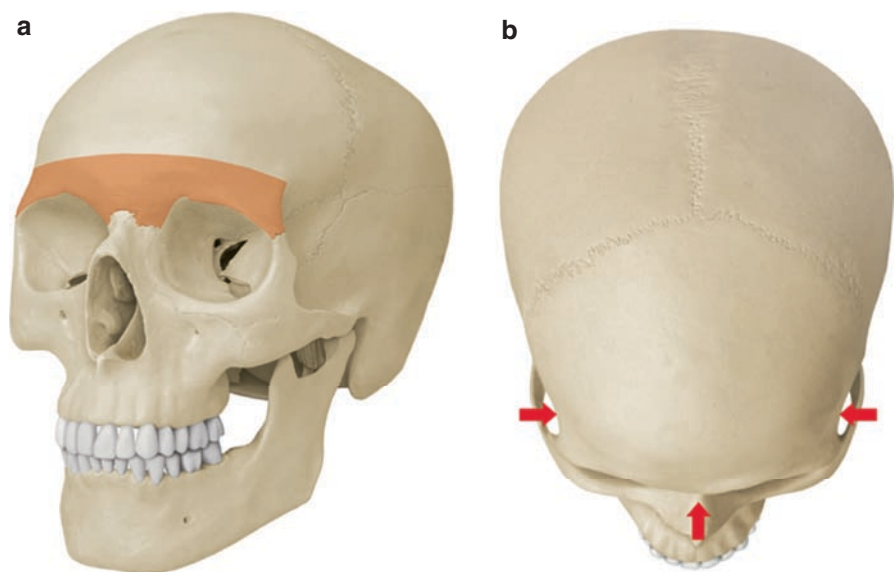
- *The position of the zygomatic arch determines the depth and the position of the zygomatic bone and the horizontal dimension of the midface*

Correct primary positioning and fixation of the zygomatic complex in relation to the skull base assures not only the correct facial width but also the correct position of the naso-ethmoidal complex. In this respect, it is the key to correct sagittal and transverse reconstruction of the midface, including vertical height. Consequently, the position of the zygomatic complex is also responsible for symmetry and ventral projection of the midface (Gruss et al. 1985 b; Gruss and Mc Kinnon 1986; Sailer and Graetz 1991; Prein et al. 1998; Manson et al. 1999).

11.2.2 Internal Midfacial Skeletal Framework

The internal skeletal frame comprises the central naso-ethmoido-orbital complex and the ventro-median struts. After reconstruction of the external frame, the successive reconstruction of the internal frame follows by progressive osteosynthesis of the central midface and its integration into the stable external frame. The reconstruction process begins in the region of the NOE complex with fixation of the central midface complex

Fig.11.3 (a) The frontofacial and the zygomatico-facial compartments are the key landmarks for the sagittal and transverse reconstructions of the midface. The frontofacial bar should be stabilized as a key lower landmark in frontal bone reconstruction. (b) The proper alignment of the zygomatico-facial compartment is the key for the correct reconstruction of the facial width and the depth of the inner orbit. The adequate reconstruction of the frontofacial compartment guarantees the correct sagittal position of the NOE complex and the zygomatico-orbital structures (mod. a. Prein et al. 1998)



to the fronto-nasal compartment. Subsequently, the infraorbital frame is connected to the central midface complex by establishing a stable longitudinal axis along the ventro-median facial struts (zygomatico-maxillary and infraorbital-nasal struts) (Gruss et al. 1985 b; Manson et al. 1985, 1987; Manson et al. 1999).

Reconstruction of the orbital walls can only be undertaken following complete reconstruction of the osseous orbital frame (Hammer and Prein 1998; Prein et al. 1998). Extremely comminuted sections of the orbital wall must be replaced by bone transplants.

- Reconstruction principles concerning the transverse and sagittal midfacial projection are based on correct primary osteosynthesis of the zygomatico-orbital and naso-ethmoidal compartments (Sailer and Grätz 1991; Prein et al. 1998)

11.3 Osteosynthesis of the Midface

Great progress in treating craniofacial fractures was made by the transition from wire osteosynthesis to rigid internal fixation in craniofacial reconstruction using different plating systems. Osteosynthesis using micro- or miniplates is today regarded as “conditio sine qua non” (Schilli 1977; Champy et al. 1978, 1986; Paulus and Hardt 1983; Stoll et al. 1985; Schwenger 1986; Jackson et al. 1986; Klotch and Gilliland 1987; Weerda and Joss 1987; Prein and Hammer 1988; Luhr 1988, 1990; Mühling and Reuter 1991; Hoffmeister and Kreusch 1991; Hausamen et al. 1995; Joss et al. 1996; Assael 1998; Prein et al. 1998; Härle et al. 1999; Eufinger et al. 1999; Greenberg and Prein 2002).

- The aim of a functional, stable osteosynthesis in an anatomically correct position is always to neutralize all forces acting on the fragments (for example, tension, pressure, and rotational forces) to prevent the fragments from dislocation (Prein et al. 1998; Härle et al. 1999)

Standardized mini- and microplates possess a high degree of ductility and permit an optimal adaptation to the thin facial bones, so enabling a precise and anatomically exact reconstruction, sustaining functionally important bone sections. The *plates and screws*, which vary in dimension, *provide a three-dimensional*

stability, which makes postoperative intermaxillary fixation superfluous (Hoffmeister and Kreusch 1991; Jensen et al. 1992; Assael 1998).

- *Stability of the frontofacial and midfacial reconstruction* protects skull base reconstruction, as the endangering mobility to the duraplasty is eliminated
- *Midfacial osteosynthesis* on the other hand, makes it possible to reconstruct the original facial dimensions — the sagittal projection, as well as the transverse (facial width) and vertical (facial height) projections — with correct occlusion

In the case of multiple strut fractures or an instable/deficient osteosynthesis, fragment dislocation against the neutralizing forces must be anticipated. This is evident by the vertical struts in midfacial elongation and by the horizontal midfacial struts in broadening of the facial skeleton (Ernst et al. 2004).

11.3.1 Plating Systems

Different osteosynthesis systems are used to reconstruct the facial skeleton according to the highly variable cranial bony structures. The systems are described according to screw diameter. All titanium osteosynthesis plates are monocortically fixed with self-cutting screws.

11.3.2 Miniplates: Microplates

- Plate thickness/form

Osteosynthesis plates should readapt fractured bony fragments in an accurate anatomical position and neutralize forces acting against the fragments (Greenberg and Prein 2002; Ernst et al. 2004). If forces are not sufficiently neutralized; the fragments are loosened and redislocated. Subsequent fissuring with the risk of fissural osteitis or pseudarthrosis may be the consequence.

Due to their flexibility, *straight plates* offered with a diverse number of holes can be modulated in all three dimensions and ideally adapted to the required demands.

Preformed plates with a variable amount of holes are inserted where plates with a strong curvature are

required, as bending with pliers results in immanent structural changes to the plate. Mesh-plates are particularly used for reconstructing comminuted fractures.

Miniplates are available in varying thicknesses, form, and screw diameter (Jackson et al. 1986; Greenberg and Prein 2002)

Miniplates – types (1.5/2.0 mm) ^a	Plate thickness 0.9 mm
Microplates – types (1.0/1.3 mm) ^a	Plate thickness 0.5 mm

^aScrew diameter

11.3.3 Screw Systems

- Screw types

Screws are subdivided according to their diameter and thread. In facial surgery, predominantly screws with a thread reaching along the total length of the screw are used. The thread runs asymmetrically to the screw core.

Screw sizes lie between 1.0- and 2.5-mm thread diameter. Emergency screws are available for the majority of standard screws, which have slightly larger dimensions. The screws are retained in the bone through friction produced by the thread (Phillips and Rahn 1989).

There are two different systems implemented in creating threads:

- A thread produced by a self-cutting screw
- A pre-cut thread produced by a screw tap

Self-cutting screws are based on the principle of the wood screw. A burr hole with a diameter slightly smaller than that of the screw is drilled into the bone and the screw is placed into it.

Self-cutting screws are differentiated into two different types:

- Thread-cutting screws
- Thread-forming screws

In the *thread-cutting screw*, where the principles of fixation and thread-cutting are combined, there are two to three slits at the tip of the screw, which collect the bone dust produced by screwing and simultaneous thread-cutting.

In the *thread-forming screw* the thread is pressed into the bone. There are no slits to collect the bone dust. Locally, high pressure strains occur, which may lead to microfractures in the surrounding bone.

The optimized construction principle in the self-cutting screws ensures maximum stability. Together with the complimentary instruments, surgery is considerably simplified and shortened (Schmoker et al. 1982; Greenberg and Prein 2002).

- Screw fixation

One differentiates between a *monocortical* and *bicortical* screw fixation. With monocortical fixation, stability is gained by penetration of only one cortical layer. The opposite cortex remains untouched. In the facial-skeletal area it is noteworthy that bicortical bone is only to be found in the mandible; this is the only significant area for bicortical fixation. Here, the outer and inner cortexes are used for stabilization.

11.4 Surgical Procedure: Osteosynthesis of the Midface

Osteosynthesis in midface fractures is orientated according to the course of the vertical and horizontal struts. Despite the fine and gracile bone structure in the mid-face, mini- and microplates can be fixed in almost any position with sufficient stability (Yaremchuk et al. 1992; Wolfe and Baker 1993). Whilst microplates are primarily used to treat fractures of the *fine nonload-bearing bone sectors* (for example, the naso-ethmoidal, infraorbital, and frontal regions), the stronger *mini-plates* are used for stabilizing the *load-bearing, vertical osseous struts* in the zygomatic and maxillary regions (Fonseca and Walker 1991; Prein et al. 1991, 1998).

11.4.1 Different Plate Sizes: Indication

As a general rule, in the region of the load-bearing trajectories 2.0-mm miniplates are used, whilst in neutral, nonload-bearing areas 1.5-mm miniplates are used. In fine nonload-bearing regions of very thin bone, 1.3-mm microplates can be applied. The thickness of the skin in delicate regions, such as the periorbital and nasal areas, should be taken into consideration.

Fracture-related plate dimensions (Greenberg and Prein 2002; Marchena and Johnson 2005; Stewart 2005a, b)

In facial reconstruction, depending on the traumatic impact and region, micro- and miniplate systems varying in dimension are applied. In the cranial compartment, micro-/miniplate systems of 1.3 or 1.5 mm are used; in the midfacial compartment, according to bone thickness, 2.0- or 1.5-mm miniplate systems are indicated in the region of the bone struts and 1.3-mm microplate systems in the orbital and naso-ethmoidal regions (Greenberg and Prein 2002).

Indications: Miniplate system, 1.5/2.0 mm

Application in

- Zygomatico-orbital fractures
- Maxillary fractures
- Zygomatico-maxillary fractures
- Zygomatico-frontal frontal
- Frontonasal fractures
- Zygomatic arch fractures
- Cranial fractures

Indications: Microplate systems, 1.0–1.3 mm

Fragments can be stabilized and fixed using these tiny plate-and-screw systems. However, one can only exceptionally succeed in neutralizing functional forces.

- Areas for application of 1.3-mm systems:
Zygomatico-orbital, cranial-fronto-glabellar, NOE, zygomatic arch, periorbital region and nasal skeleton, NOE fractures, cranial fractures
- Areas for application of 1.0-mm systems:
Very fine osseous areas such as the maxillary sinus wall, infraorbital margin, nasal bone, alveolar process; anchoring naso-orbital-ethmoidal and small cranial fragments

11.4.2 Fracture-Related Osteosynthesis

There are standardized principles concerning the osteosynthesis of anatomically correct repositioned skeletal structures. The osseous struts and trajectories define the areas for an elective, stable osteosynthesis according to their thickness (Champy et al. 1978, 1986; Klotch and Gilliland 1987; Prein et al. 1998; Härle

et al. 1999; Booth et al. 2003; Ernst et al. 2004). The principle of stability requires that the osteosynthesis plate must be fixed to a stable, immobile midfacial structure with a minimum of two screws.

11.4.2.1 Surgical Approaches

The approach in *central pyramidal fractures* (Le Fort II, Wassmund II fractures) is carried out by a combination of an intraoral vestibular incision with an extraoral transconjunctival, subciliary, infraorbital, or mediopalpebral approach. In selected cases, a median nasofrontal, vertical approach may be necessary. Alternatively, a midfacial degloving through intraoral and intranasal incisions can be performed (Thumfart et al. 1998).

In *centrolateral fractures* (Le Fort III, Wassmund IV fractures), a combination of an intraoral, vestibular approach with a mediopalpebral and supraorbital or/and lateral approach may be sufficient for exposure (Brisett and Hilger 2005). In case of extensive *zygomatic bone dislocations or complex NOE fractures*, the coronal incision is the approach of choice for fracture treatment (Perott 1991) (Fig. 11.4).

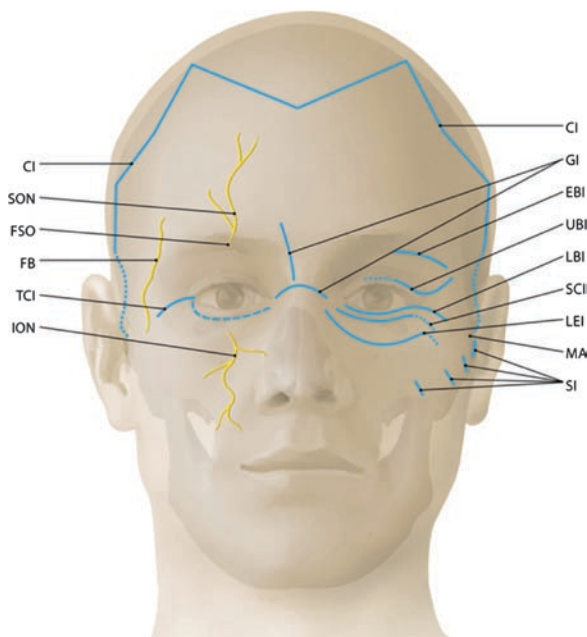


Fig.11.4 Anterior surgical approaches to the facial skeleton (mod. a. Prein et al. 1998, Prein and Lüscher 1998). CI coronal incision, GI glabellar incision, SCI subciliary incision, TCI transconjunctival incision, LBI lower blepharoplasty incision, LEI lower eyelid incision, UBI upper blepharoplasty incision

11.4.2.2 Lateral Midface Fractures

The amount of osteosynthesis required depends on the degree of dislocation and the extent of the fracture in the midfacial skeleton (Rowe 1985; Chuong and Kaban 1986; Jackson 1989; Zingg et al. 1991; Ellis, 1991; Prein et al. 1998; Gruss et al. 1999; Maniglia et al. 1999; Stewart 2005 b; Marchena and Johnson 2005).

- **One-point plate fixation:**
Fixation at the zygomatico-frontal suture for stabilization of nondislocated zygoma fractures. Osteosynthesis using supraorbital-lateral 2.0-mm miniplates (Prein et al. 1998; Markowitz and Manson 1998).
- **Two-point plate fixation:**
Fixation at the zygomatico-frontal suture and at the zygomatico-alveolar crest or infraorbital for stabilization of dislocated zygoma fractures with a rotational component. Osteosynthesis using supraorbital-lateral 2.0- or 1.5-mm miniplate and infraorbital 1.3-mm microplates (Prein et al. 1998; Markowitz and Manson 1998).

- **Three-point plate fixation:**
Lateral midface fractures with distinct dislocation but secured sagittal projection are stabilized using three-plate osteosynthesis at the zygomatico-frontal suture with 2.0-mm miniplates, infraorbital margin with 1.3-mm microplates and the zygomatico-alveolar crest with 1.5- or 2.0-mm miniplates (Holmes and Matthews 1989; Prein et al. 1998; Markowitz and Manson 1998) (Fig. 11.5–11.7).

- **Four-point plate fixation:**
In cases of extensive comminution with the loss of sagittal projection, there is a selective necessity to expose and stabilize the zygomatic arch. Osteosynthesis is performed using 1.5-mm miniplates. Occasionally, depending on extent and localization of the fracture region, further additional osteosynthetic stabilization is necessary in the cranial, maxillary or/and facial region (Fig. 11.8–11.12).

- **Concomitant orbital floor/orbital wall fractures**

It is obligatory to expose, control, and reconstruct the orbital floor in cases of complex or dislocated

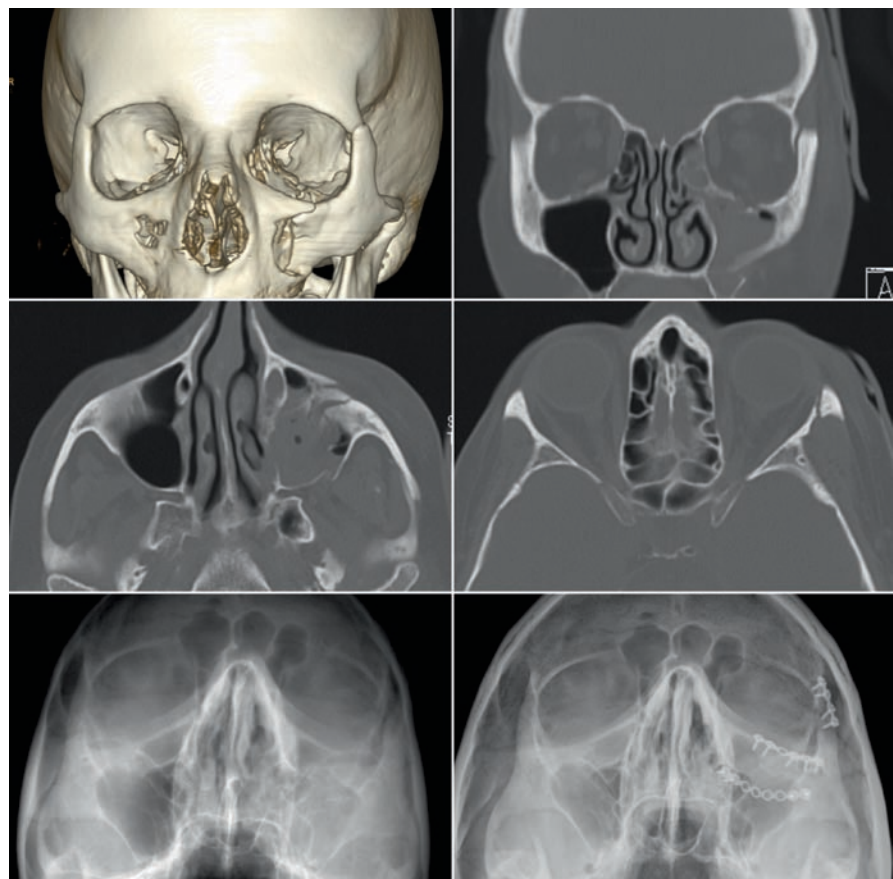


Fig.11.5 Reposition and fixation of a lateral midface fracture with three miniplates at the lateral orbital wall, infraorbital rim and anterior wall of the maxillary sinus

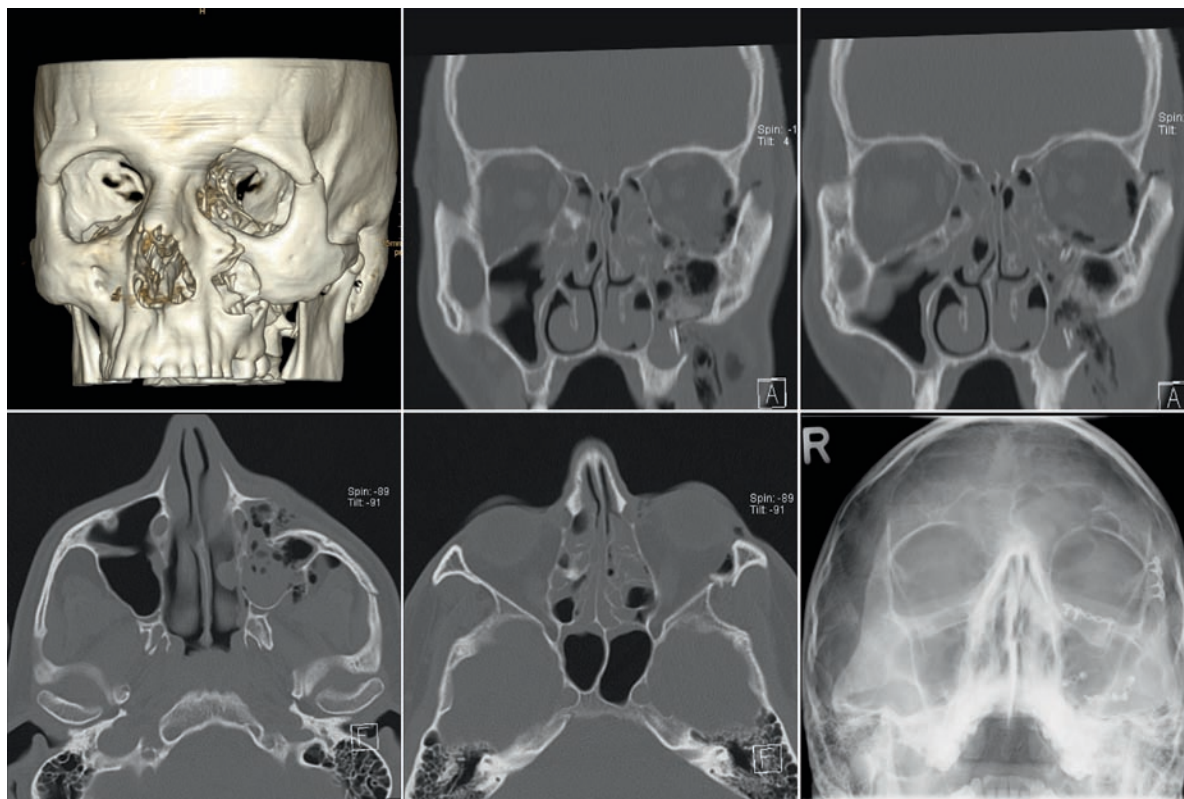


Fig.11.6 Reposition and fixation of a complex lateral midface fracture with significant displacement of the zygomatic bone into the maxillary sinus with three mini-plates: at the lateral

orbital wall, infraorbital rim and zygomatico-maxillary buttress. Emphysema in the orbit and pterygomandibular fossa

zygomatico-orbital fractures, particularly in the case of verifiable dislocated orbital floor fractures with or without functional deficits (Chen et al. 1992; Joos 1995; Brady et al. 2001; Stewart and Soparkar 2005).

Access to the orbital floor is normally achieved via a subciliary, a medio-palpebral or a transconjunctival incision (Manson et al. 1987; Hammer 1995, 2001). In general, the same rules apply to treating orbital wall fractures (Jackson et al. 1986; Serletti and Manson 1992; Hammer and Prein 1998) (Fig. 11.13–11.15).

- Removal of the dislocated fragments and repositioning of the periorbital. In the case of medial orbital wall fractures, simultaneous treatment of the fractured ethmoid cells.
- Reconstruction of the orbital walls by repositioning the bony fragments; small gaps are covered with a patch. In extensive defects, reconstruction of the orbital walls with autogenous grafts or alloplastic membranes is necessary (Fukado et al. 1981).

- Transplant fixation in the orbital floor or medial/lateral orbital wall is carried out with microscrews or plates (Frodel 2002).

In complex orbital fractures, the stronger periorbital bone segments are first repositioned and stabilized by miniplate osteosynthesis before the fragile structures of the orbital walls are reconstructed (Stewart 2005 b; Marchena and Johnson 2005).

11.4.2.3 Central: Centrolateral Midface Fractures

The following principles apply to central subcranial and centrolateral midface fractures (Schwenzer 1986; Prein et al 1998; Manson 1998; Marchena and Johnson 2005; Stewart 2005a, b):

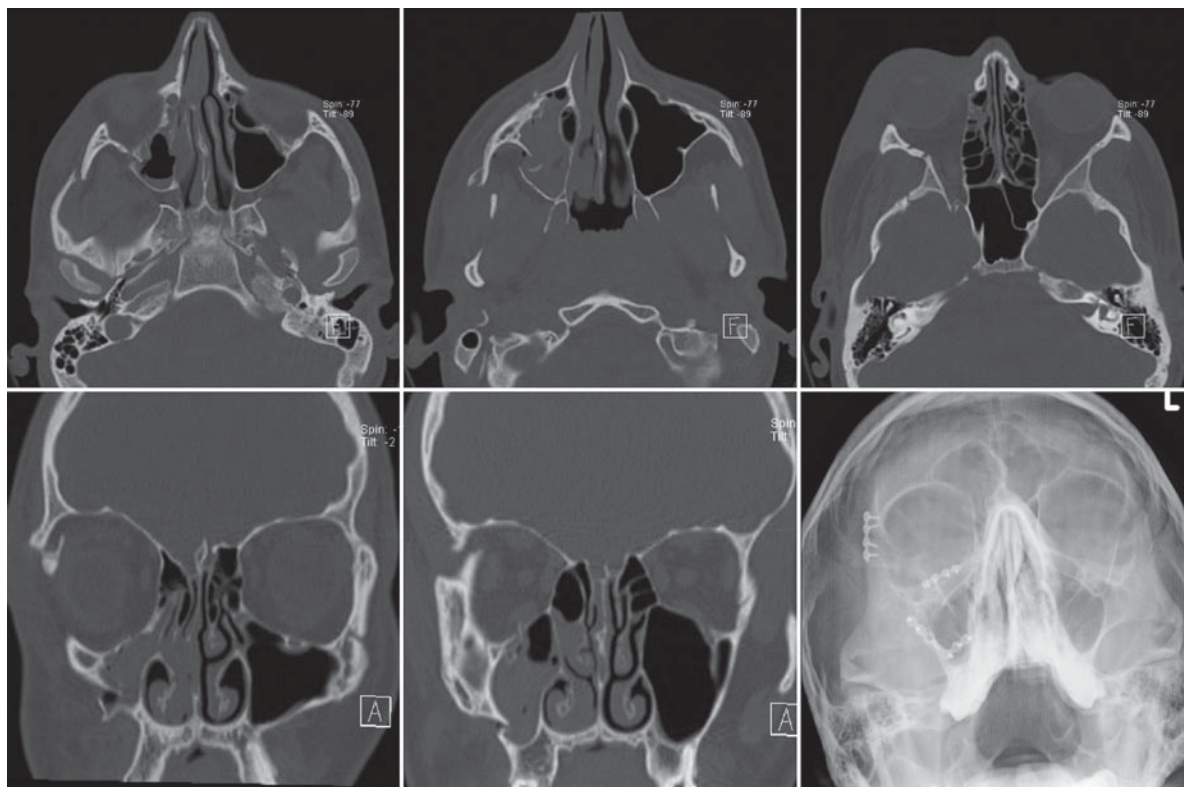


Fig.11.7 Posterior displacement of the zygomatic bone, fracture of the anterior and lateral maxillary sinus walls, fracture of the zygomatic arch and outward displacement of the lateral

orbital wall. Reposition and fixation with three mini-plates at the lateral orbital wall, infraorbital rim and zygomatico-maxillary buttress

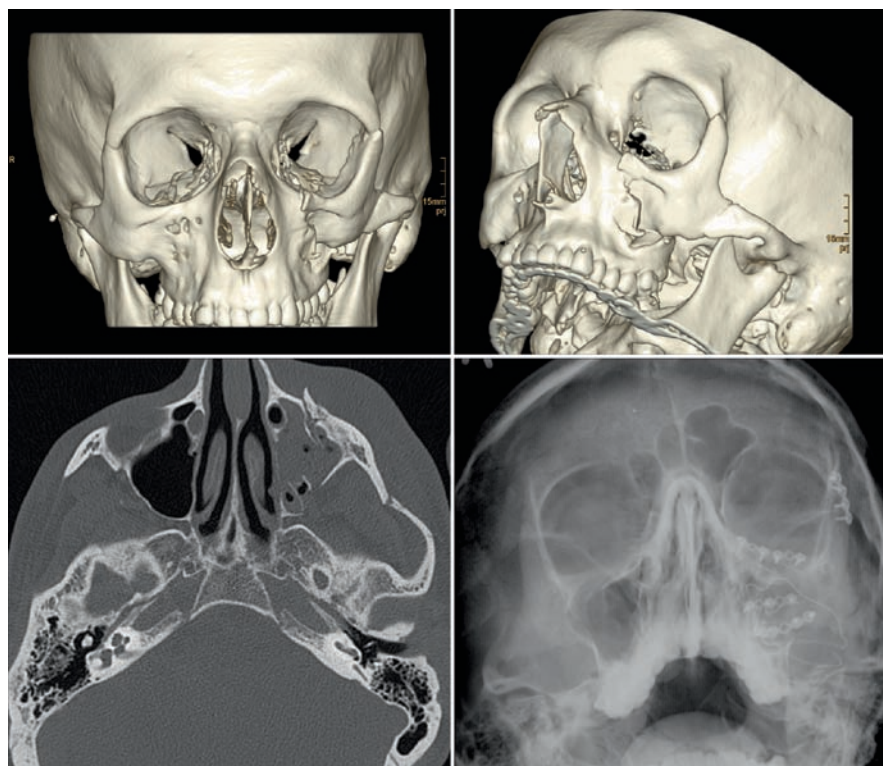


Fig.11.8 Reposition and fixation of a moderately depressed lateral midface fracture with four mini-plates at the lateral orbital wall, infraorbital rim, zygomatico-maxillary buttress and anterior maxillary sinus wall

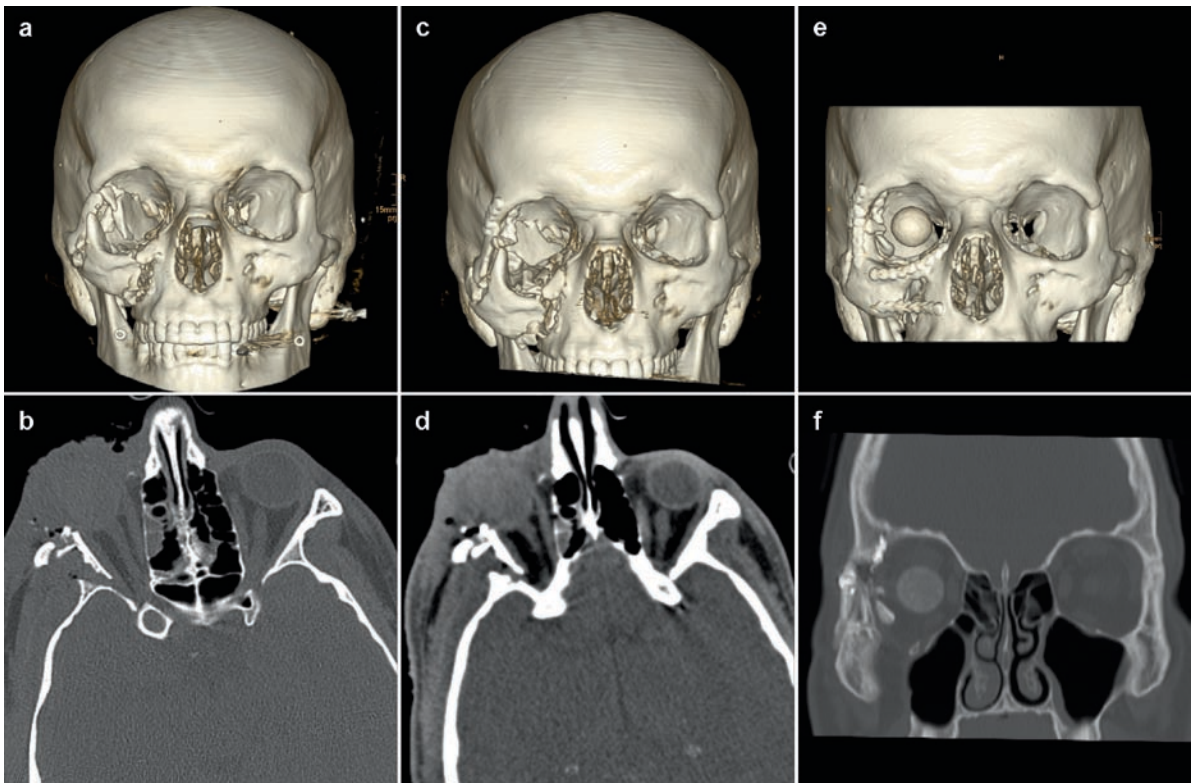


Fig.11.9 Reconstruction of a complex lateral orbito-zygomatic fracture with extrusion of the globe (a,b,d,e). Decompression of the orbit with reposition of the malrotated zygomatic bone and

osteosynthesis of the lateral orbital wall, the inferior orbital rim and anterior maxillary wall (c). The destroyed globe was removed and replaced with an epithesis (c, f)

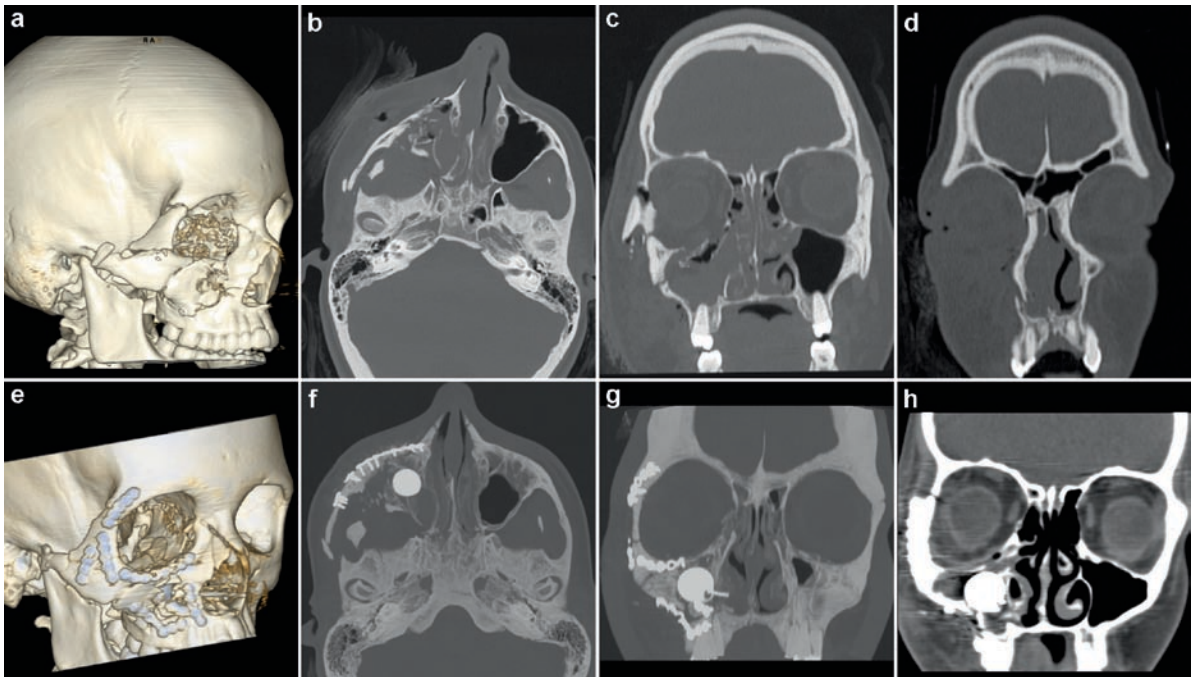
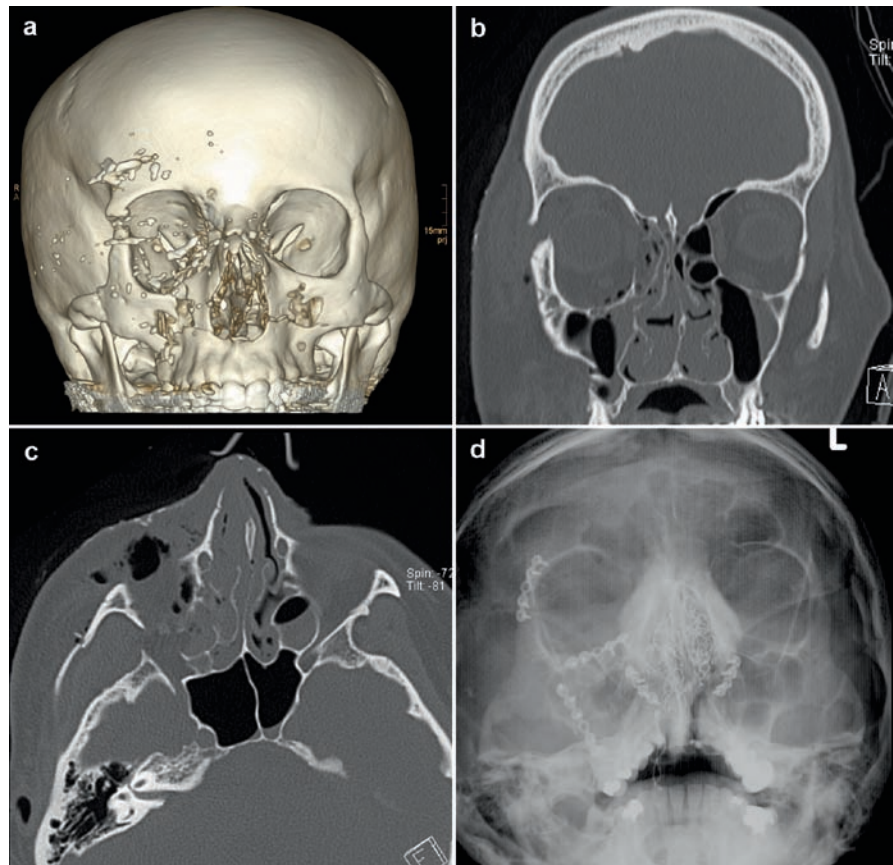


Fig.11.10 Fragmentation of the zygomatic bone with flattening of the lateral midface and deconfiguration of the orbit. (a–d) Complex fracture of the lateral orbital wall and orbital floor (c). Application of multiple osteosynthesis plates (7) (e–h). PDS foil

implant to support the orbital floor and prevent soft tissue herniation into the fracture defects (f, g, h). Stabilization of the anterior maxillary walls and the volume of the sinus with a Foley catheter (f, g)

Fig.11.11 Stabilization of a lateral midface fracture combined with bilateral Le Fort-I-fracture of the maxilla with five miniplates.

(a) Multiple foreign bodies (glass fragments) in the skin.
(b, c) Bilateral Le Fort-I-fracture, dislocated zygomatico-orbital fracture and blow-out fracture of the medial orbital wall right.
(d) Postoperative radiograph: three plates for stabilization of the lateral midface fracture, two paranasal plates to fixate the maxilla. Tamponade in the nasal cavity



- Le Fort I fractures

Classically Le Fort I fractures are treated by osteosynthesis in the region of the anterior nasal aperture (apertura piriformis) and the zygomatico-alveolar buttress using four 2.0-mm miniplates (Stanley 1990).

Smaller bone fragments from the antral region are reintegrated and fixed with 1.3-mm miniplates. Additional sagittal maxillary fractures are stabilized with a transversal plate below the piriform aperture and occasionally by an additional transversal palatal 1.5-mm miniplate (Sofferman et al. 1983; Manson et al. 1990; Gruss and Philipps 1992; Prein et al. 1998; Härle et al. 1999; Stewart 2005a; Marchena and Johnson 2005).

Reconstruction of the anterior wall of the maxillary sinus is carried out by reintegrating the bony fragments or with bone grafts, but also by applying titanium meshes to avoid soft tissue collapse or invasion into the sinus (Kuttenberger and Hardt 2001; Marchena and Johnson 2005; Stewart 2005a, b).

- Le Fort II fractures/Wassmund II fractures

Le Fort II fractures are stabilized in the region of the naso-frontal suture, the zygomatico-alveolar buttress and the infraorbital margin. Following instrumental repositioning (ROWE- forceps) and intermaxillary adjustment to the position of central occlusion, the Le Fort II complex is bilaterally fixed to the zygomatico-alveolar buttress with 1.5-mm miniplates and to the infraorbital region with 1.3-mm miniplates. A facultative fixation to the naso-frontal region using 1.5-mm miniplates may be necessary (four-point osteosynthesis) (Prein et al. 1998).

Remaining bony fragments are reintegrated using 1.5-mm or 1.3-mm mini- or microplates. Alternatively, the orbital floor may either be reconstructed with autogenous fragments, autogenous bone grafts, alloplastic membranes or titanium-mesh systems.

- Le Fort III fractures/Wassmund III and IV fractures

Due to its vertical pull, the masseter muscle occasionally causes extensive caudal dislocation. Osteosynthesis

Fig.11.12 Coronal approach for placement of multiple osteosynthesis plates for stabilization of a severely displaced comminuted centro-lateral midface and zygomatic arch fracture

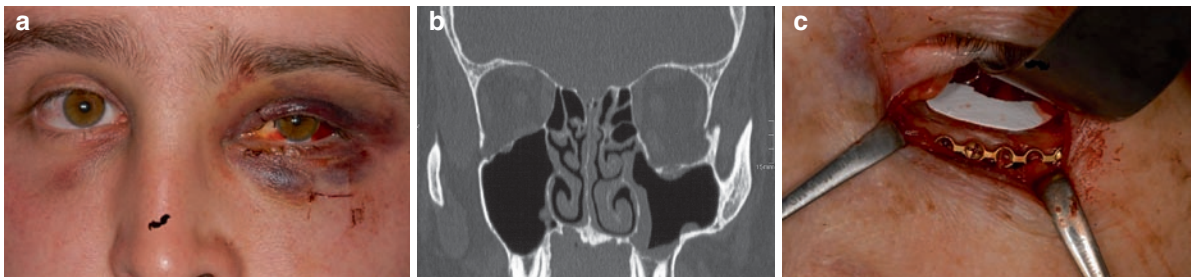
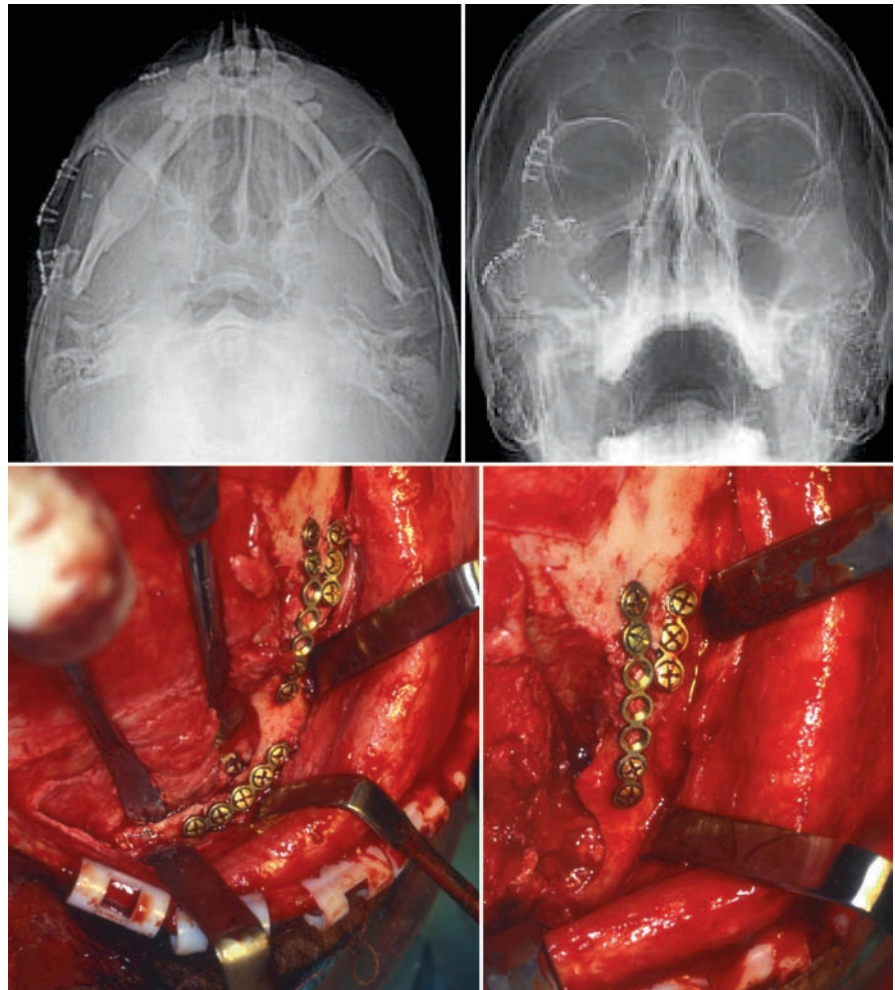


Fig.11.13 Surgical reconstruction of the orbital floor (intraoperative views). (a) Herniation of orbital fat into the maxillary sinus. (b) Defect of the orbital floor after reposition of the fatty-

orbital tissue (arrow). (c) Reconstruction of the orbital floor using polyurethane foil (Neuropatch)

Fig.11.14 CT-based intraoperative guidance for reconstruction of the medial orbital wall. (a) Three-planar virtual view for intraoperative navigation indicating surgical position. (b) Transnasal endoscopic view after reconstruction of the medial wall with PDS foil (arrow) from a transfacial-orbital approach

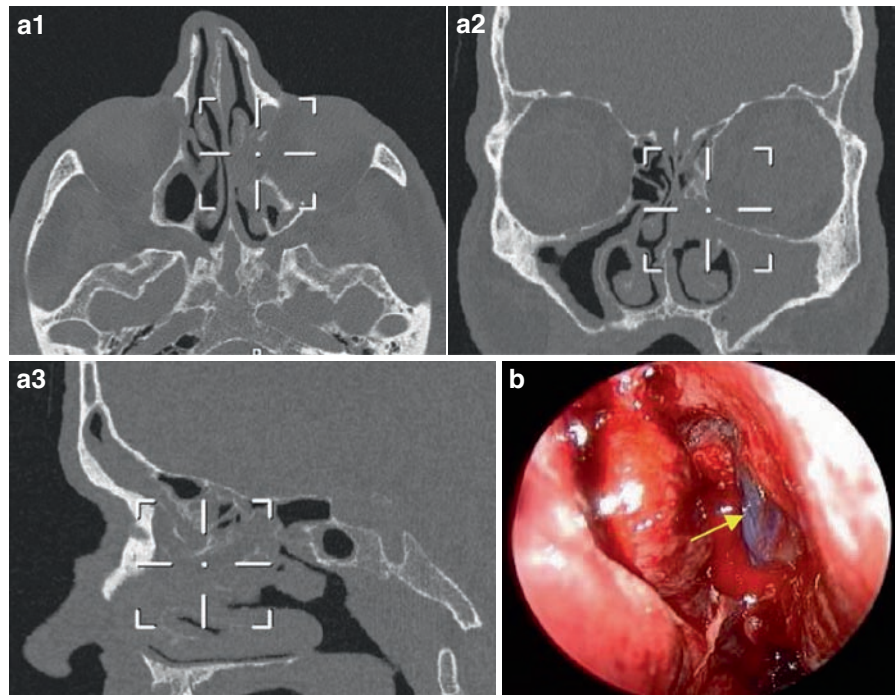
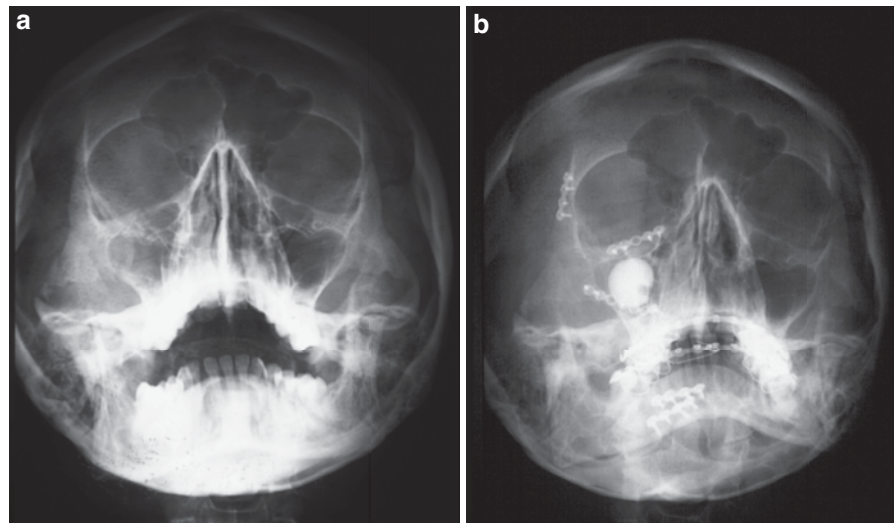


Fig.11.15 Reconstruction of a displaced right zygomatico-orbital fracture (a) with three miniplates and repositioning of the orbital floor fragments. Additional antral balloon stabilization and osteosynthesis of a concomitant paramedian mandibular fracture on the right (b) (pre-/postoperative)



must, therefore, be able to withstand tensile forces, especially in the region of the zygomatico-frontal suture and naso-frontal suture.

Following manual or instrumental repositioning and intermaxillary fixation in a position of central occlusion, osteosynthesis is carried out bilaterally at the zygomatico-frontal suture using 2.0-mm miniplates and optionally at the naso-frontal suture with 1.5-mm miniplates (Prein et al 1998; Manson 1998).

- Le Fort III fractures and Wassmund II fractures

Combined Le Fort III/Wassmund II fractures are stabilized at the naso-frontal suture (1.5-mm miniplates), at the region of the infraorbital margin (1.3-mm microplates), the zygomatico-frontal suture (2.0-mm miniplates) and the zygomatico-alveolar buttress (1.5-mm miniplates) (Prein et al. 1998; Manson 1998).

11.4.2.4 Complex Midfacial Fractures

- Subcranial midfacial fractures

In the majority of cases, combined midfacial fractures (complex midface fractures/complex maxillo-mandibular fractures) have irregular fracture courses. Therefore, depending on their course, these fractures often require osteosynthesis with multiple 1.3-, 1.5-, and 2.0-mm miniplates in the region of both the vertical and horizontal struts. Bony fragments are stabilized with 1.3-mm microplates. Autogenous bone grafts are necessary in case of structural deficits (Manson 1986; Markowitz and Manson 1998; Gruss et al. 1990; Manson et al. 1995, 1999; Prein et al. 1998; Marchena and Johnson 2005; Stewart 2005a; McGraw-Wall 2005) (Figs. 11.16 and 11.17).

Procedure (complex midface fractures) (Gruss and Philipps 1989; Manson et al. 1990; Prein et al. 1998)

- Fracture exposition:
 - At the zygomatico-maxillary and naso-maxillary struts through an intraoral-vestibular incision
 - At the infraorbital margin through bilateral subciliary or medio-palpebral incision
 - At the zygomatico-frontal suture through a supraorbital-lateral incision
 - Optional supplementary medio-nasal or coronal incision (fracture: NOE complex)
- Mobilization and repositioning of the maxillary segments and intermaxillary fixation
- Reposition of small bony fragments in the region of the vertical struts (reestablishment of maxillary and midfacial vertical height)
- Zygomatico-maxillary, naso-maxillary, infraorbital, zygomatico-frontal, and fronto-nasal osteosynthesis. Optional osteosynthesis to bridge the gaps between fractured osseous struts is achieved with osteosynthesis plates or bone grafts to prevent midfacial collapse
- Reconstruction of orbital floor/orbital wall defects using bony fragments, bone grafts, membranes or microtitanium meshes

- Central upper midface fractures — NOE fractures

Following a coronal or case-related individual approach and repositioning of the naso-ethmoidal complex

(Markowitz and Manson 1998; Markowitz et al. 1991; Mathog 1992, 1995; Gehrke et al. 1996; Kessler and Hardt 1998; Oeltjen and Hollier 2005; Stewart 2005a,) stabilization is performed along the naso-frontal suture using 1.5-mm miniplates. Paranasal and infraorbital osteosynthesis of the lateral naso-frontal and naso-maxillary bone fragments is performed with 1.3-mm microplates as well as interfragmental stabilization (Leipziger and Manson 1992; Mathog et al. 1995). Displaced naso-orbital bone fragments with an attached medial palpebral ligament are fixed using microplates and transversal, intercanthal wire osteosynthesis (Hammer and Prein 1998) (Fig. 11.18).

Naso-Ethmoidal (NOE) Fractures: Type 1

Following open transfacial “en bloc” reduction, type 1 NOE fractures are fixed along the fronto-maxillary strut in the region of the anterior bony aperture of the nose, in the fronto-nasal region and at the infraorbital margin using 1.5/1.3-mm mini-/microplates (Ellis 1993; Mathog et al. 1995; Prein et al. 1998; Donald 1998).

Naso-ethmoidal (NOE) Fractures: Types 2 and 3

In fracture type 2 with multifragmental injuries to the NOE compartment and loss of intercanthal ligament insertion, correct *osseous reconstruction of the naso-orbital structures* is mandatory. Following reduction of bony fragments and provisional, interfragmental wire osteosynthesis, the NOE complex is then definitely fixed to the surrounding, stable midface structures, beginning in the region of the medial orbital border, superiorly to the cranial and caudally to the maxillary complex using 1.5/1.3-mm mini-/microplates (Prein et al. 1998).

Osteosynthesis of small fragments in the medial orbital region is carried out using 1.0/1.3-mm microplates (Markowitz et al. 1991; Leipziger and Manson 1992; Ellis 1993; Prein et al. 1998). However, 2.0-mm miniplates are not suitable because of the delicate soft tissues in the medial orbital region (medial and anterior to the lacrimal fossa) (Hammer and Prein 1998). An additional direct transnasal wire-fixation of the ligament-bearing fragments avoids dislocation of the reduced fragments at a later date (Figs. 11.19 and 11.20).

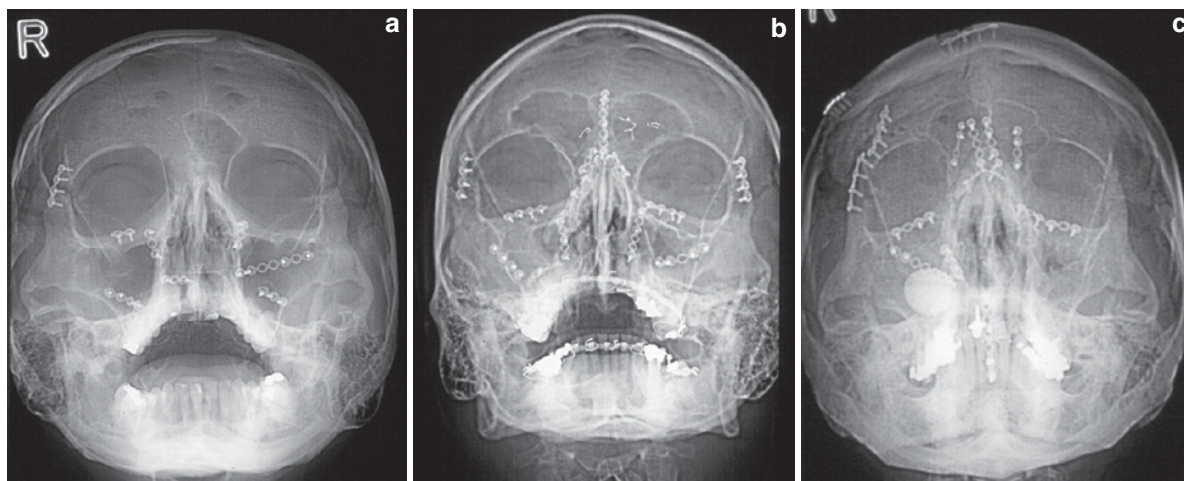


Fig.11.16 Examples of osteosynthesis with multiple 2.0/1.5-mm mini-plates of three different types of subcranial fractures (CMF and CUMF). (a) Internal fixation in a CMF fracture. (b) Internal

fixation in a CCMF without frontobasal fracture. (c) Internal fixation in a CUMF with frontobasal fracture

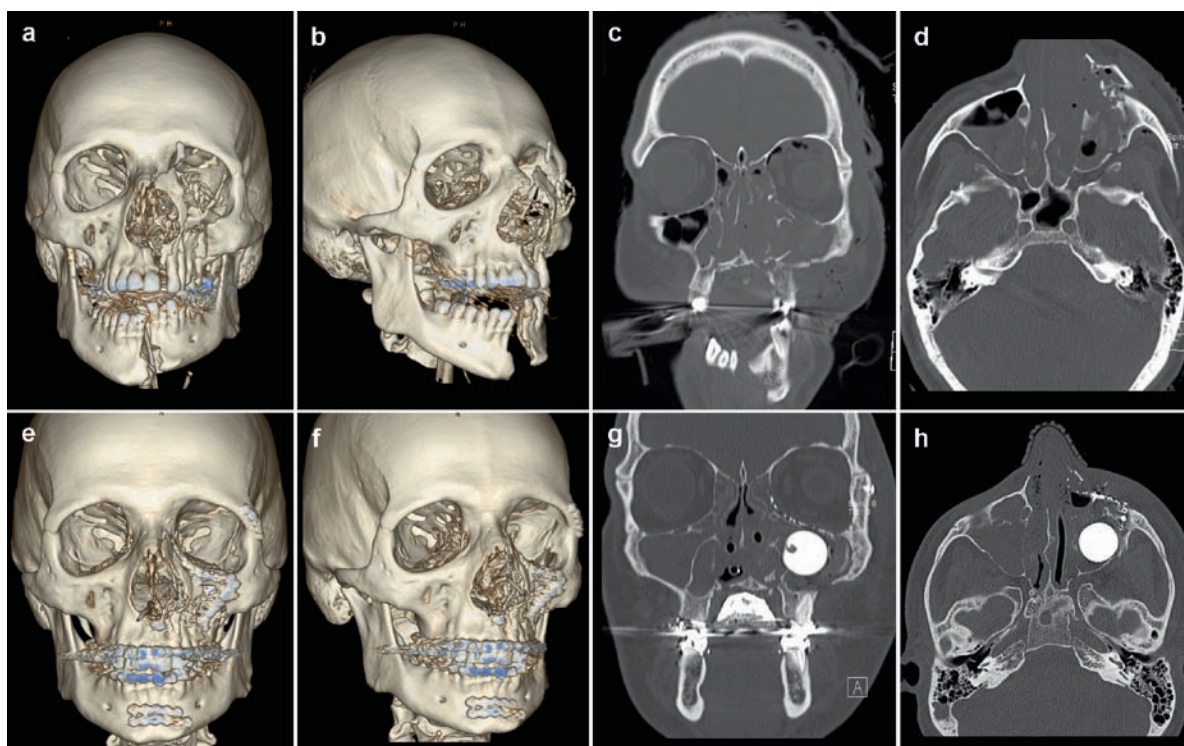
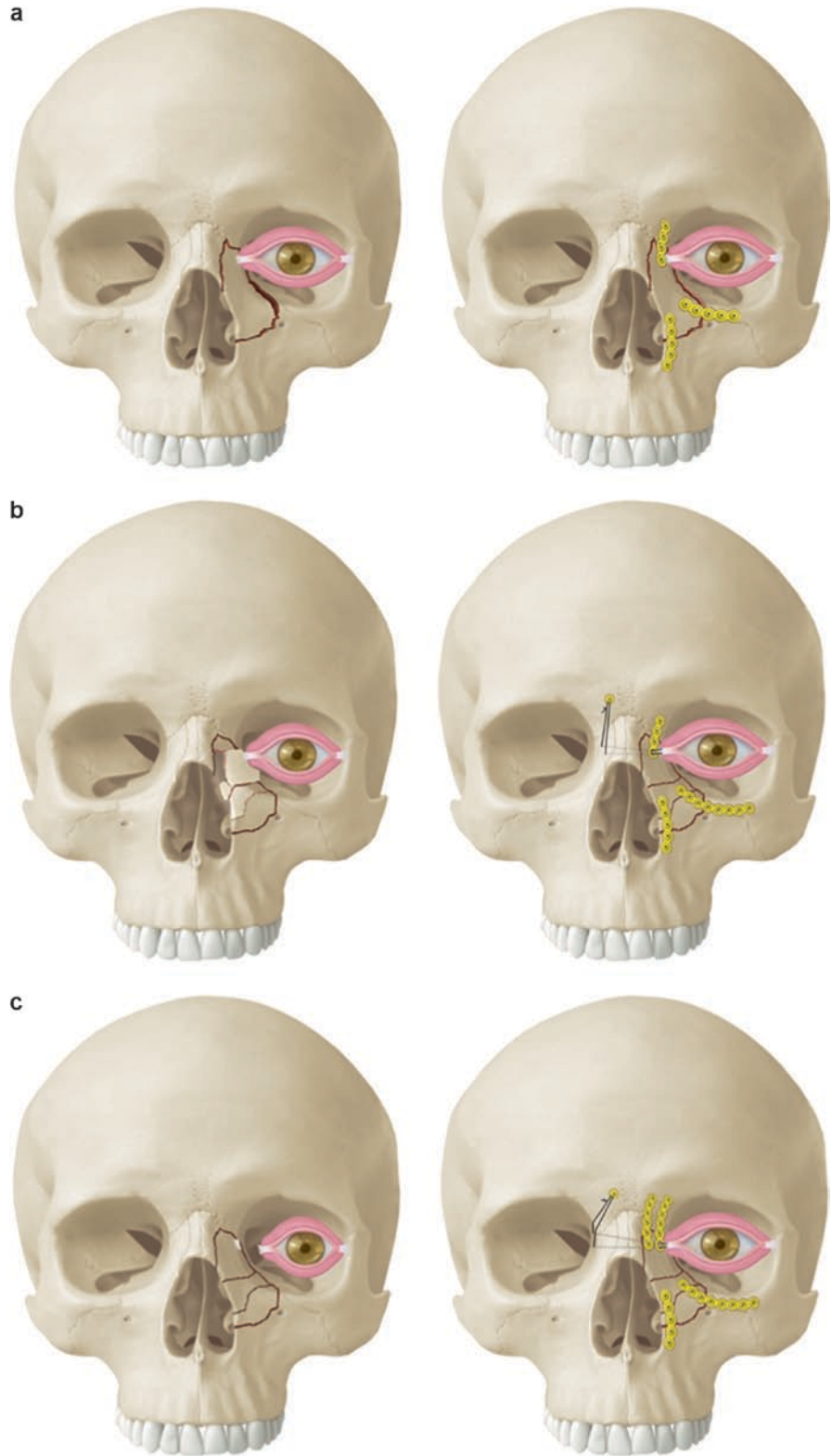


Fig.11.17 Gun shot trauma with burst fracture of the maxilla and mandible. (Submental gun position). (a, b, c, d) Internal osteosynthesis with multiple 2.0-mm miniplates (e, f). Postoperative control demonstrating reconstruction of the left maxillo-zygomatic-orbital

complex and the mandible. The reconstruction of the orbital floor and anterior wall of the maxillary sinus was performed with micro-titanium mesh. Antral balloon catheter for hemostasis (g, h)

Fig.11.18 Classification and treatment of different NOE fractures (mod. a. Prein et al. 1998, Hammer und Prein 1998). **(a)** Type-1 NOE fracture. Stabilization of a single large fragment with 1.5- and 1.3-mm miniplate systems. **(b)** Type-2 NOE fracture. Stabilization with combined 1.3-mm plates and transnasal wire through the fragment bearing the canthal ligament. **(c)** Type-3 NOE fracture. Stabilization with 1.3 mini- or 1.0-mm microplates. Refixation of the canthal ligament by direct transnasal canthopexy. A missing insertion point may be recreated by a bone graft or a miniplate. The typical location is posterior and superior to the lacrimal fossa



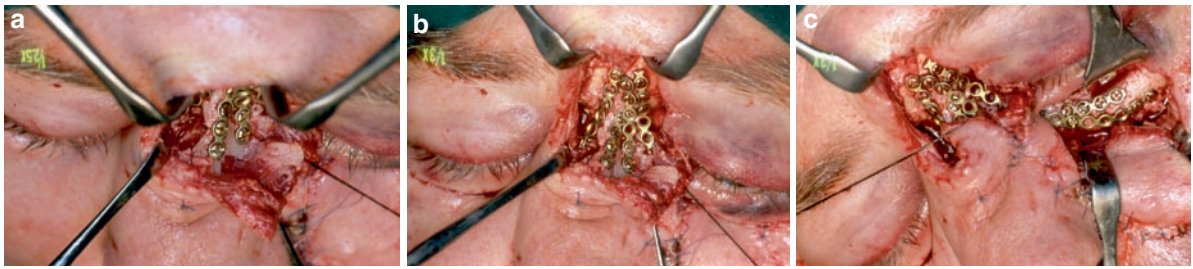


Fig.11.19 Internal osteosynthesis in a comminuted NOE fracture (type 2) with frontoglabellar-paranasal and infraorbital osteosynthesis (1.5-mm miniplates) using the transfacial approach

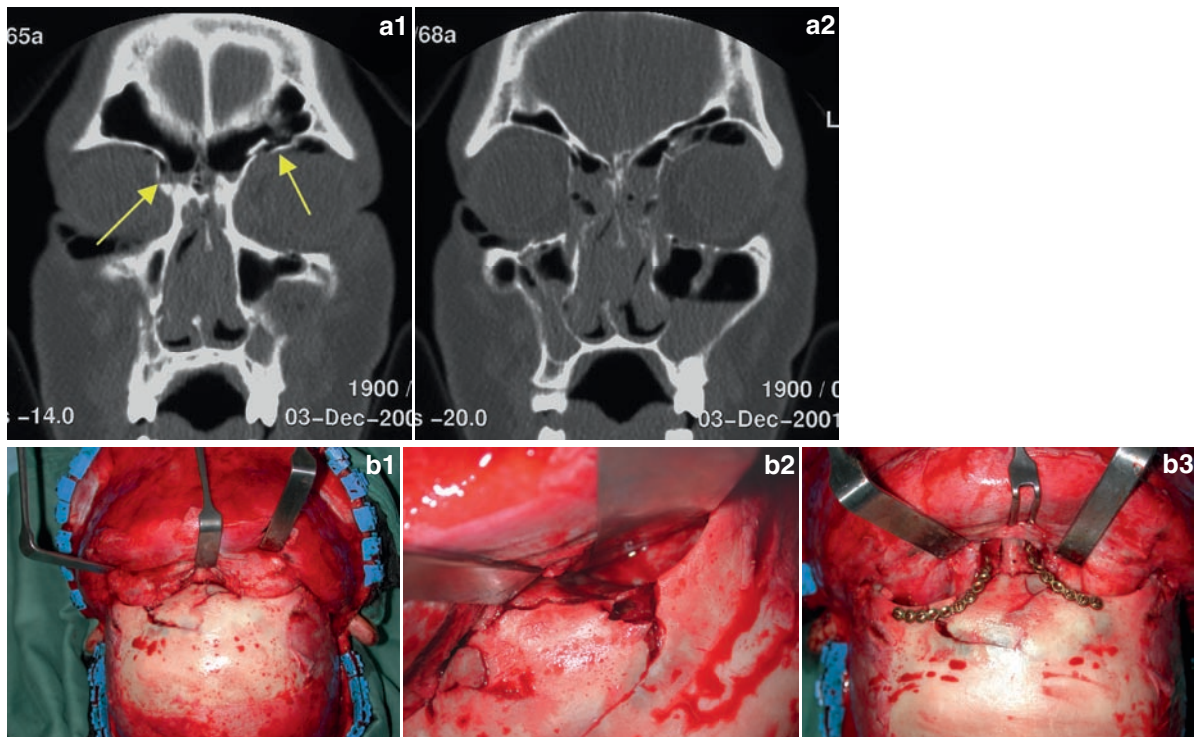


Fig.11.20 Upper midface fracture (CUMF: central midface, NOE and anterior sinus wall fracture). (a) CT scan demonstrating fractures in the upper medial quadrant of the orbit (arrows).

(b) Exposition and reconstruction of the fronto-naso-maxillary complex using the coronal approach

Canthal ligament insertion (see Chap.12.2)

• Combined cranial and midface fractures

In extensive craniofacial fractures of the types comminuted upper midface fractures (CUMF) and comminuted cranio-maxillary fractures (CCMF)/comminuted panfacial fractures (PF), the skull base-related frontofacial and zygomatico-orbital structures must be accurately reconstructed prior to midface reconstruction (Gruss et al. 1989).

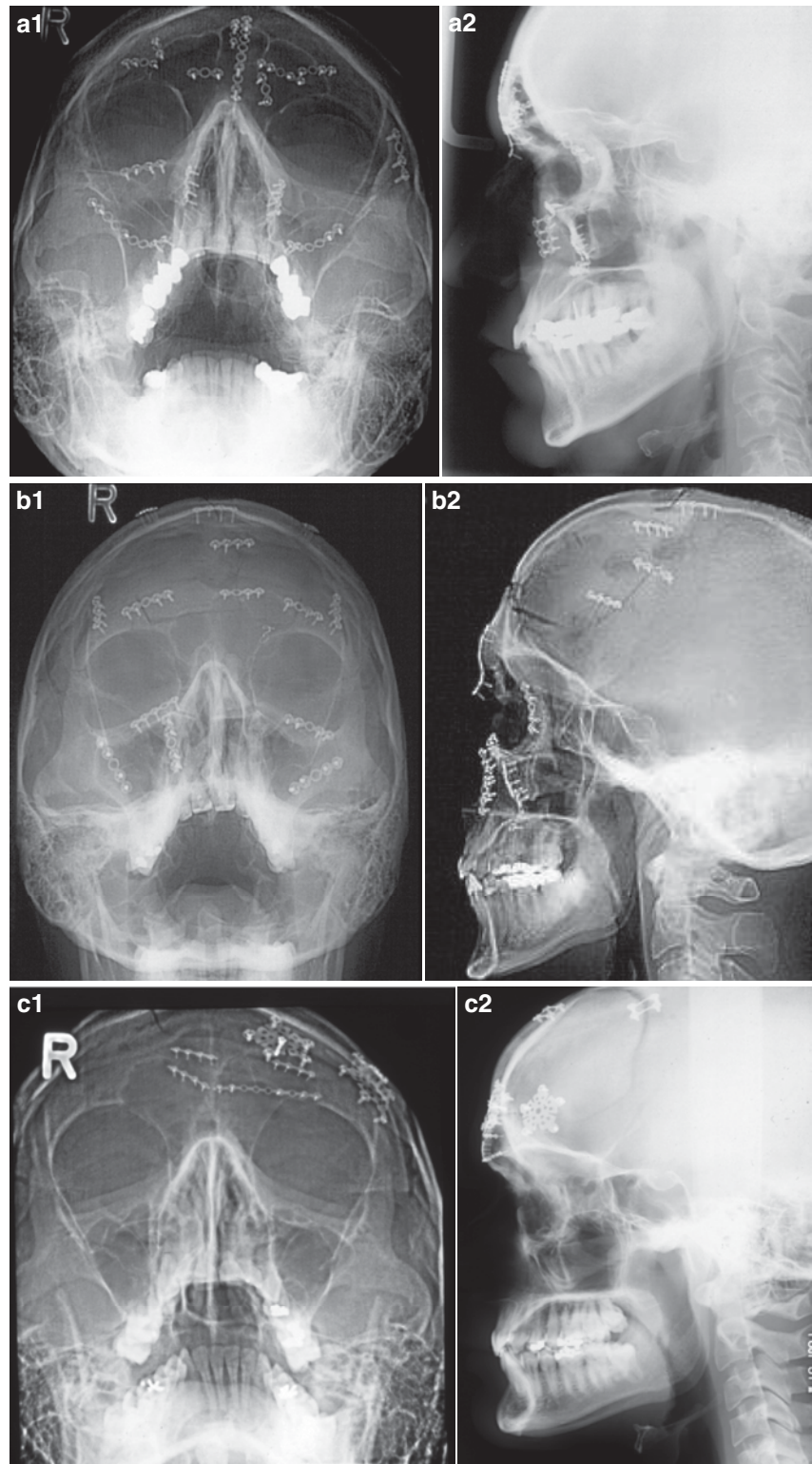
The *frontofacial and glabellar bone fragments* are stabilized with 1.5-mm miniplates. In aesthetically

important regions such as the naso-frontal region, 1.3-mm microplates are applied. Smaller frontal fragments are reintegrated and stabilized with 1.3/1.0-mm microplates.

For stability reasons, the subsequent osteosynthesis of the reduced *zygomatico-orbital complex* is performed as follows (Figs. 11.21 and 11.22):

- Supraorbital-lateral with 2.0-mm miniplates
- Zygomatic arch and infraorbital with 1.5-mm miniplates
- For reconstruction of the maxillary complex, the above mentioned criteria apply

Fig.11.21 Internal fixation of different types of comminuted craniofacial fractures (CCMF and COF) with 2.0/1.5-mm mini-plates. (a) CCMF without frontobasal fracture and dural laceration. (b) CCMF with frontobasal fracture and dural laceration. Additional craniotomy and dural reconstruction. (c) Unilateral cranio-orbital fracture (COF) with frontobasal fracture and dural laceration. Additional craniotomy and dural reconstruction



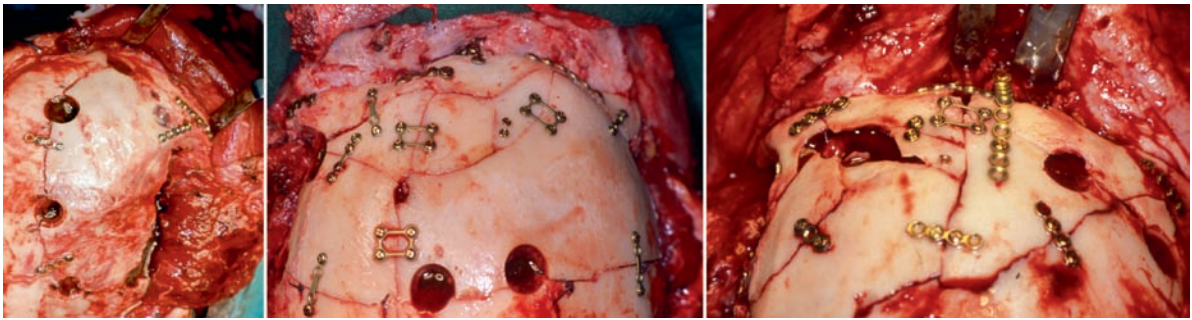


Fig.11.22 Three examples of integration and osteosynthesis of cranial bone flaps and interfragmentary fixation of multiple bone fragments with miniplates in CCMF fractures

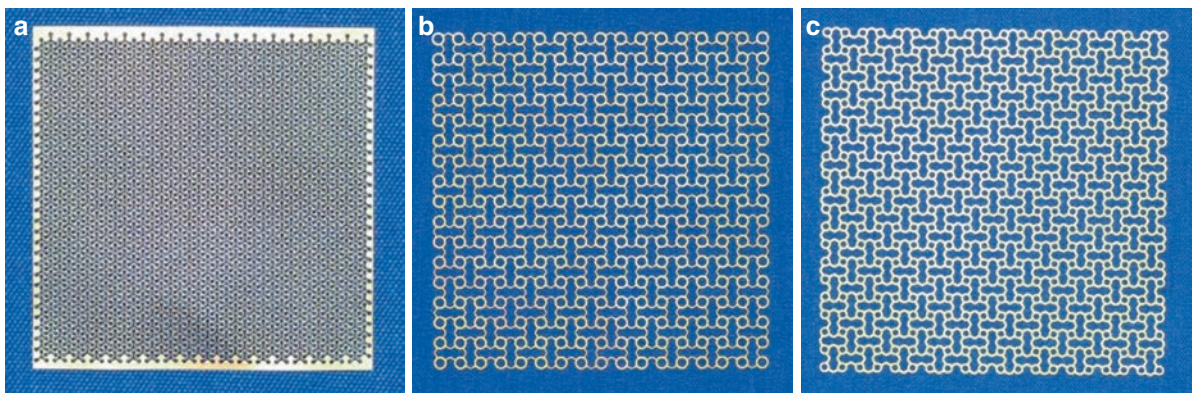


Fig.11.23 Various mesh types: dynamic titanium mesh (1, 0.3 mm; 2, 0.6 mm), microdynamic titanium mesh (3, 0.1 mm)

11.5 Titanium: Mesh-Systems

11.5.1 Mesh-Systems

The excellent biocompatibility of titanium, the unique geometrical properties of titanium meshes and their easy handling make these mesh systems an ideal substitute for treating defects in nonload-bearing areas of the skull (Arx et al. 1995; Kessler and Hardt 1996; Kuttenger and Hardt 2001).

- Dynamic mesh

(Hardt et al. 1992,1994; Reinert and Gellrich 1997; Kessler and Hardt 1996; Kuttenger et al. 1999; Kuttenger and Hardt 2001).

The dynamic mesh is available in three thicknesses: 0.1, 0.3, and 0.6 mm. Its preangled bars, which prevent kinking or wrinkling of the mesh, allow complex three-dimensional contouring, which is especially applicable for orbital reconstruction.

Stable functional and aesthetic long-term results can be achieved in this anatomically complex region. The mesh may, therefore, be an alternative to bone or cartilage grafts (Fig. 11.23).

- Microdynamic mesh

(Hardt et al. 1992,1994; Kessler and Hardt 1996; Kuttenger et al. 1999; Kuttenger and Hardt 2001).

The laser-perforated microdynamic mesh consists of pure titanium (thickness of 0.1 mm) can be cut with scissors and is easily adapted and fixed with titanium microscrews.

11.5.2 Indications and Advantages

In general, the two microtitanium mesh systems are used in the following indications:

- Immediate reconstruction of bony defects
- Correction of extensive contour irregularities in combination with bone or cartilage grafts
- Reconstruction in areas predisposed to infection with or without autogenous transplants
- Contour reconstruction in aesthetically critical regions (supraorbital margin)

In specific cases of craniofacial reconstruction, these systems offer valuable, additional therapeutic options and have several advantages (Kuttenberger et al. 1999):

Advantages

- Immediate availability
- Universal applicability (orbital walls, cranial defects, comminuted fractures in fine-walled bone areas, wall defects of the maxillary sinus)
- No donor side morbidity
- Possible three-dimensional reconstruction of complex anatomical bone structures
- Combination with bone or cartilage grafts (Reinert and Gellrich 1991)
- No memory effect, no resetting
- Extremely low infection rate
- Combination with microvascular transplants (Eufinger et al. 1999)

11.5.3 Defect Treatment Using Titanium-Mesh

Bridging defects with titanium meshes to maintain contours provides aesthetically and functionally acceptable results (Figs. 11.24–11.28).

- Minor bony defects up to a size of 25 cm² in comminuted fracture sites of nonload-bearing areas can easily be covered with the 0.3-mm mesh (Hardt et al. 1992; Kuttenberger and Hardt 2001).
- For major bone defects exceeding >25 cm², reconstruction with calvarial bone is the treatment of choice (Mohr et al. 1994). Large defects covered by split calvarial grafts can be optimized by an additional coverage with titanium mesh to create an aesthetically acceptable contour, so that conspicuous bone loss due to resorption will not become obvious (Hardt et al. 1994). Contour irregularities in the contact zones between grafts can be avoided using microtitanium mesh strips (Reinert and Gellrich 1991; Hardt et al. 1994).
- In craniofacial reconstruction, contact with the paranasal sinuses cannot be avoided. If titanium meshes are in contact with the ethmoid or the frontal sinus, re-pneumatization of the sinus normally takes place and there are almost no mesh-related infections.
- Postoperative endoscopic controls by Deinsberger et al. (1998) showed complete epithelization of

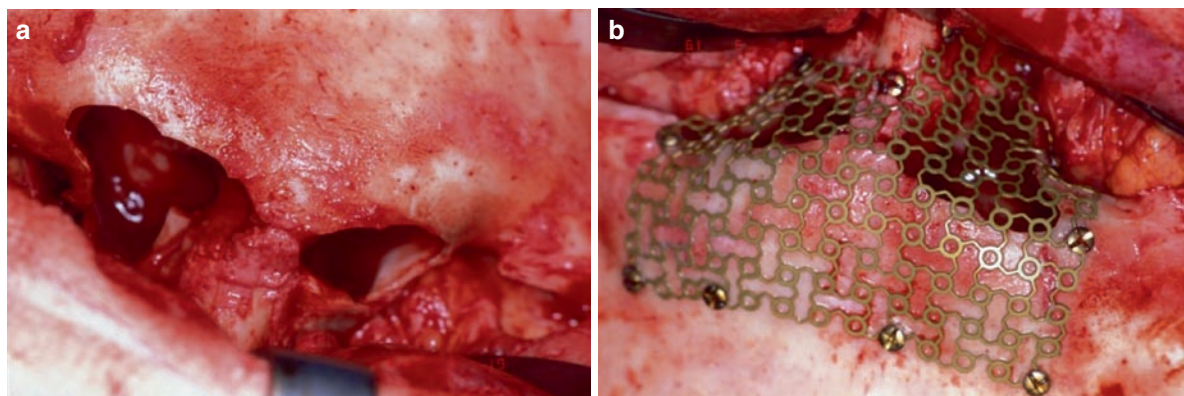


Fig.11.24 Cranial reconstruction with titanium mesh. (a) Bilateral reconstruction of the supraorbital rim and the anterior wall of the frontal sinus with dynamic titanium mesh (0.3 mm)

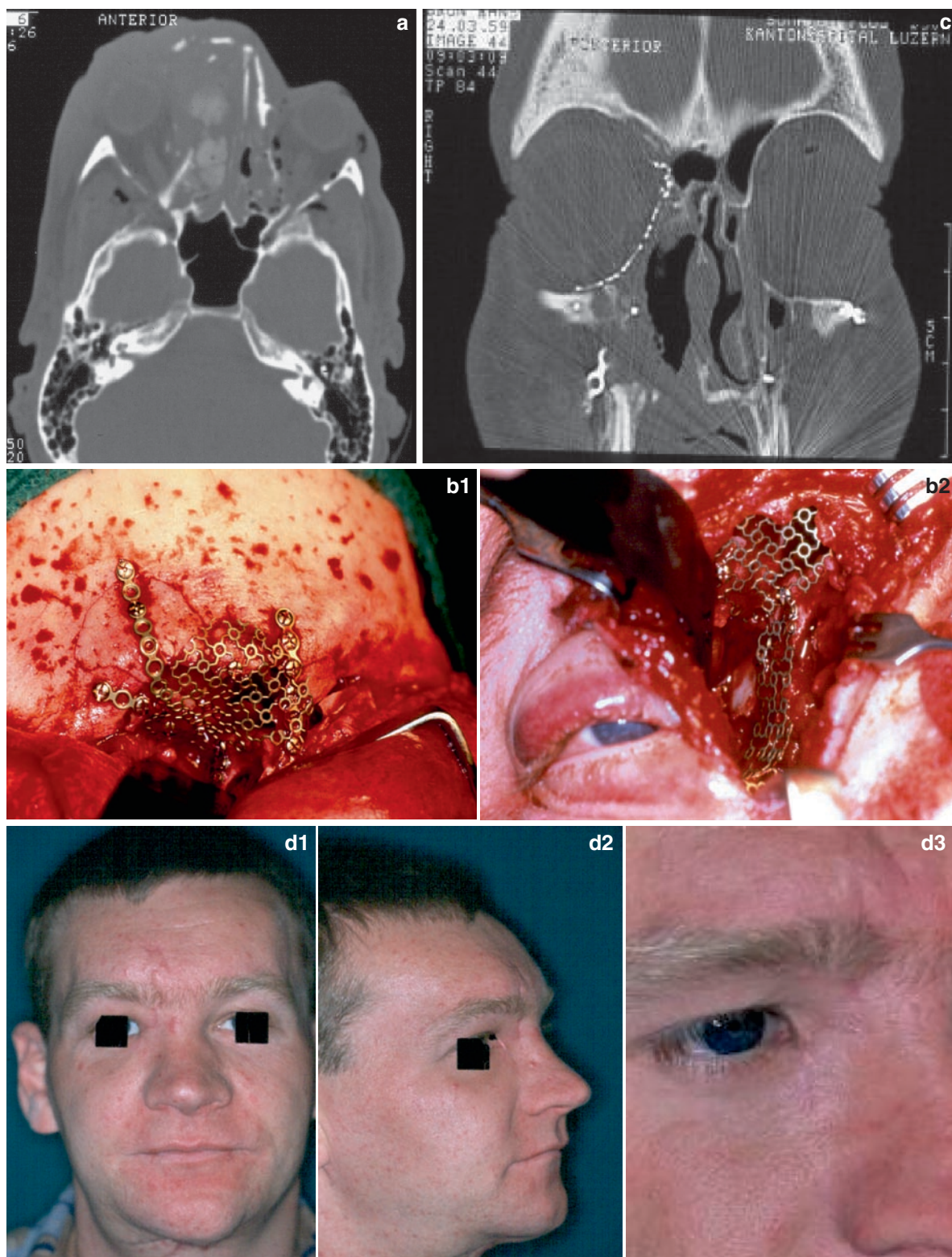


Fig.11.25 Severe maxillary-orbital and cranial defects after a submento-orbito-frontal gunshot injury. (a) Preoperative CT scan demonstrating severe comminution of the right nasal, ethmoidal, and orbital structures with extensive bone loss. (b) Reconstruction of the anterior sinus wall, supraorbital rim,

medial orbital wall and medial part of the orbital floor with dynamic titanium mesh (0.3 mm). (c) Postoperative CT scan demonstrating symmetrical reconstruction of the right orbital cavity. (d) Aesthetic result 1 year postoperatively with some inconspicuous scars

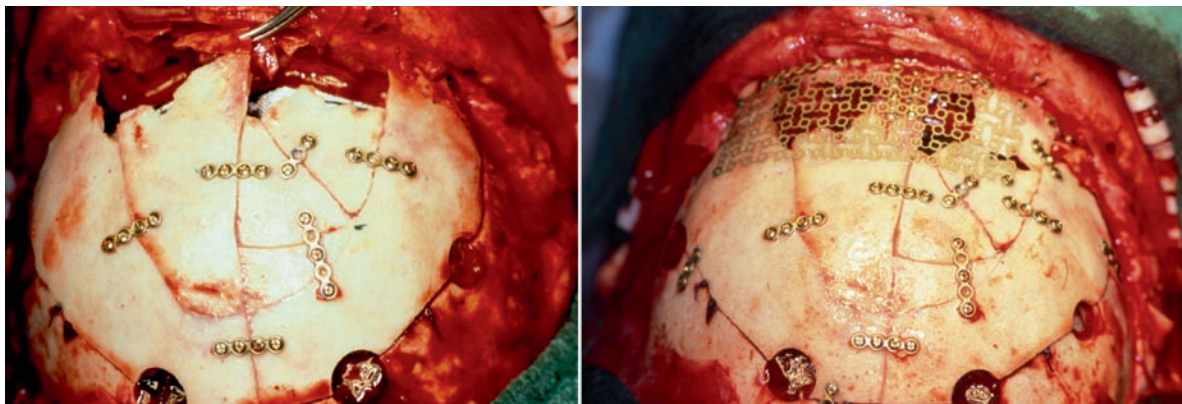


Fig.11.26 Fronto-glabellar reconstruction following craniotomy, dural repair and cranialization of the frontal sinus. Residual bony defects are covered with dynamic titanium mesh (0.3 mm)

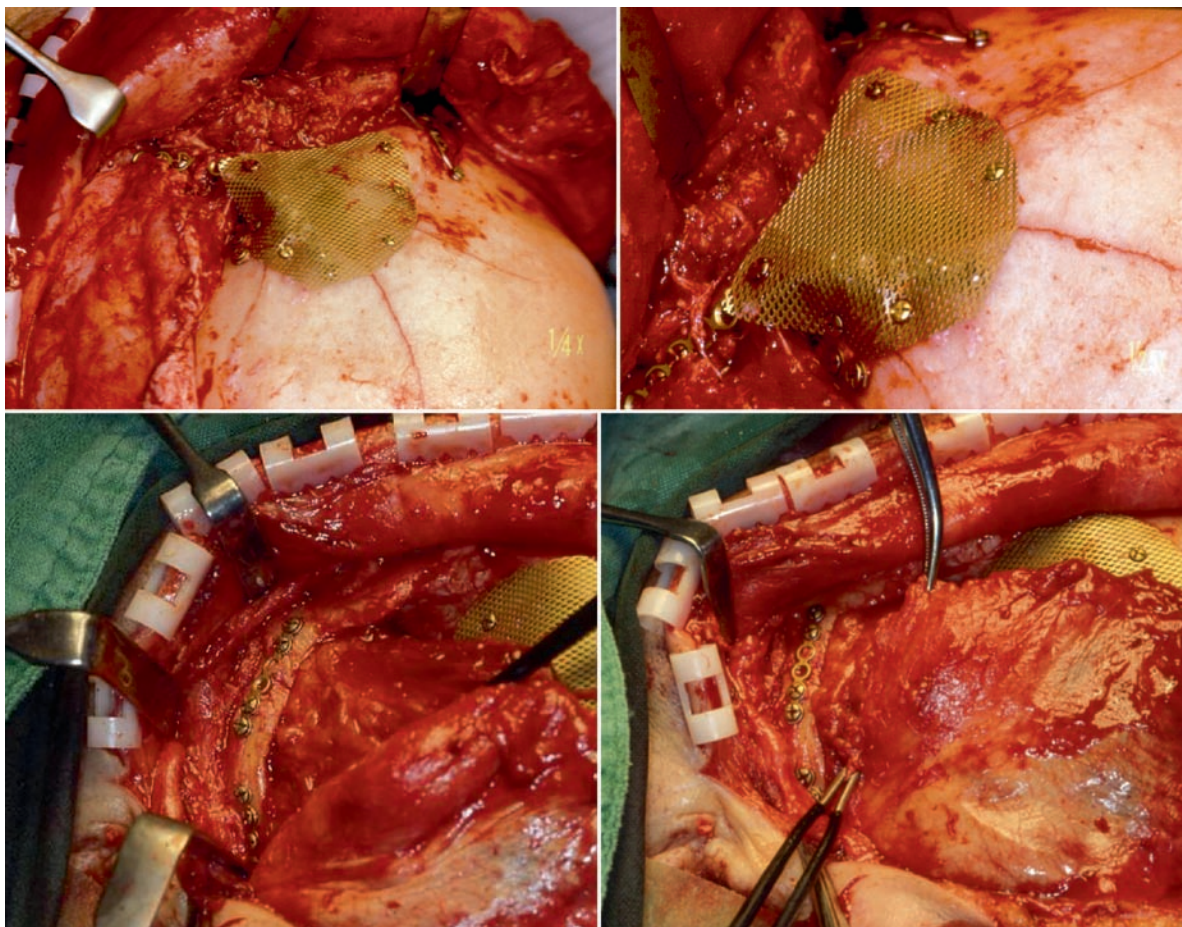


Fig.11.27 Reconstruction of the zygomatico-temporal area. After fixation of the zygomatic bone and zygomatic arch, a residual latero-temporal bone defect is covered with microtitanium

mesh (0.1 mm). The mobilized temporal muscle is attached to the mesh

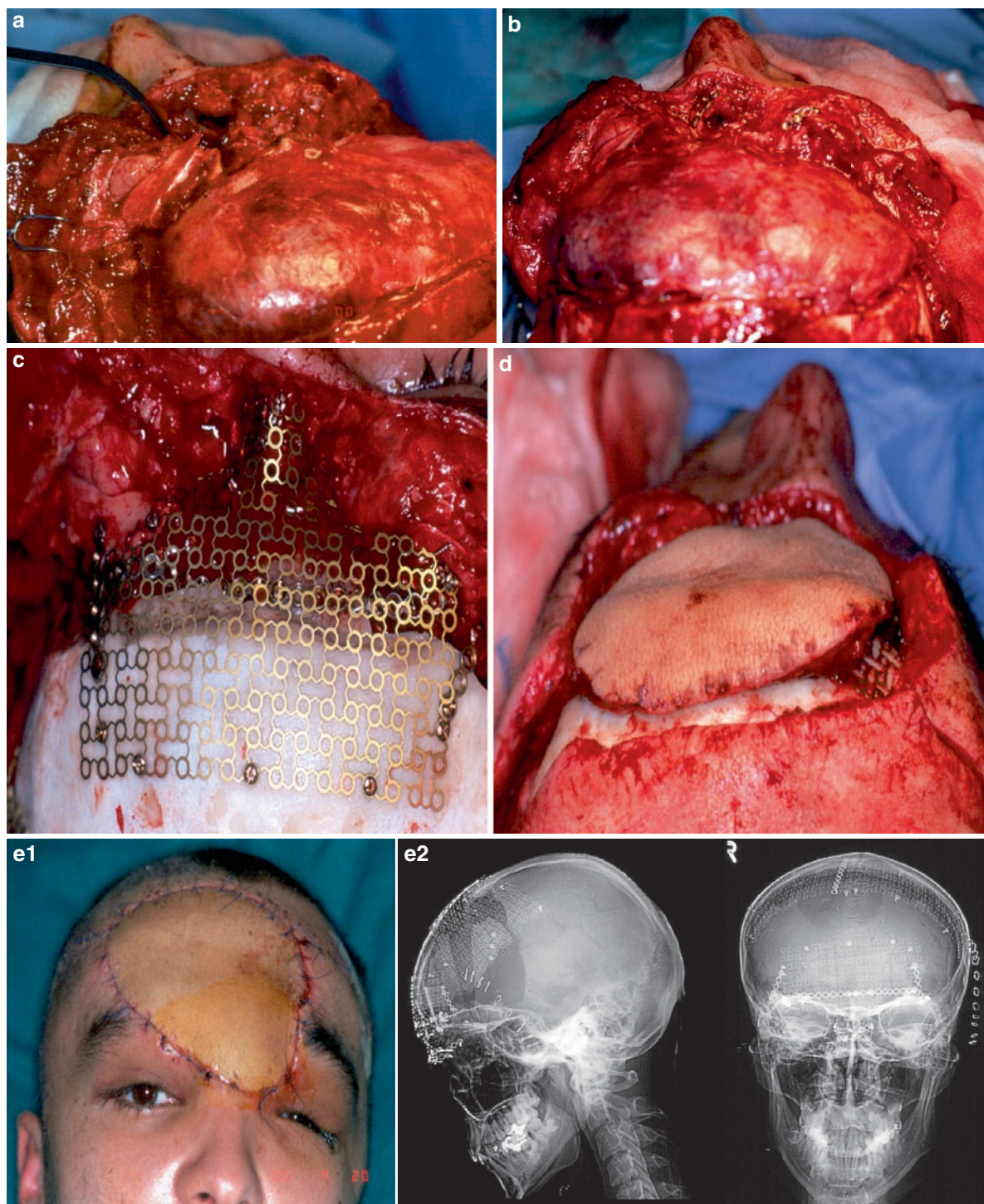


Fig.11.28 Microvascular reconstruction after extensive bone and soft tissue loss. **(a)** Avulsion injury with partial loss of dura, frontal bone and skin caused by an axe blow. **(b)** Intraoperative view after craniotomy, refixation of the naso-frontal fragments and closure of the dural defect. **(c)** The remaining bony defect is

covered with a dynamic titanium mesh (0.3 mm). **(d)** The missing frontal soft tissues are reconstructed using a radial forearm flap. **(e)** Postoperative clinical and radiological results (1 week postoperatively)

the titanium mesh on the side facing the paranasal sinuses. No clinical problems were reported through contact between the titanium mesh and the dura (Kuttenberger and Hardt 2001).

- Spontaneous neo-osteogenesis under the titanium mesh, however, cannot be demonstrated either radiologically or clinically (Hardt et al. 1994; Kuttenberger et al. 1999; Kuttenberger and Hardt 2001).
- Care should be taken not to allow the mesh to exceed the natural bony boundaries, as the mesh may then cause soft tissue irritation or perforation. In this case, it is recommended to remove the mesh partly or completely.

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12.1 Craniofacial Fractures

As a rule, severe combined skull base and facial injuries are high-velocity traumas, which often result in multi-fragmental fractures of the skull base with intracranial dislocation of bone fragments, dural tears with subsequent liquorrhea and brain tissue herniation. Extensive intracranial injuries and disfiguring facial fractures often lead to severe functional deficiencies.

Therapeutically, a considerable modification has taken place in the surgical treatment of complex craniofacial fractures. Today, an explicitly reconstructive approach in order to preserve all anatomical structures is favored (Sailer and Gratz 1991; Joos and Gilsbach 1991; Raveh et al. 1992; Hardt et al. 1992; Ewers et al. 1995; Giuliani et al. 1997; Vesper et al. 1998):

- By consequently using the transfronto-cranial, transfronto-subcranial and transfacial approaches
- By systematically stabilizing the facial skeleton using mini- and microplate osteosynthesis
- By primarily replacing missing bone with autogenous bone grafts (Jackson et al. 1986)

12.1.1 Concept of Reconstruction

In principle, the reconstructive approach in craniofacial fractures is subdivided into the following steps (Gruss 1986; Joos et al. 1989; Schilli and Joos 1991; Joos and Gilsbach 1991; Krafft et al. 1991; Rohrich and Shewmake 1992; Weingart et al. 1996; Moskopp and Horch 1996; Seidl et al. 1998; Prein 1998; Joss et al. 2001):

1. Reconstruction and stabilization of the skull base and frontofacial compartment and treatment of cerebral and dural injuries

The sequence of reconstruction is systematic. After exposing the fronto-cranial structures and the skull base, the frontofacial bandeau is reconstructed as a contouring connecting link between the neuro- and viscerocranium. Subsequently, the neurosurgical treatment of cerebral and dural injuries, reconstruction of the skull base and frontofacial reconstruction are carried out (Rohrich and Shewmake 1992; Gruss et al. 1992; Schierle and Hausamen 1997).

Sequence of reconstruction (Schierle and Hausamen 1997)

- Transfrontal or transfacial fracture exposition
- Exploration of the frontofacial compartment
- Exploration of the skull base
- Stabilization of the fronto-cranial fragments
- Treatment of dural and cerebral injuries, skull base reconstruction

If these basic principles are not followed, there is a considerable risk of unsatisfactory bony reconstruction, recurrent liquorrhea and functional impairment (Machtens 1987) (Fig. 12.1).

2. Reconstruction and stabilization of the midface compartment

If midface reconstruction and stabilization is not correctly performed, it may lead to facial shortening if there is insufficient vertical stabilization and facial widening as a result of insufficient reconstruction in the horizontal dimension.

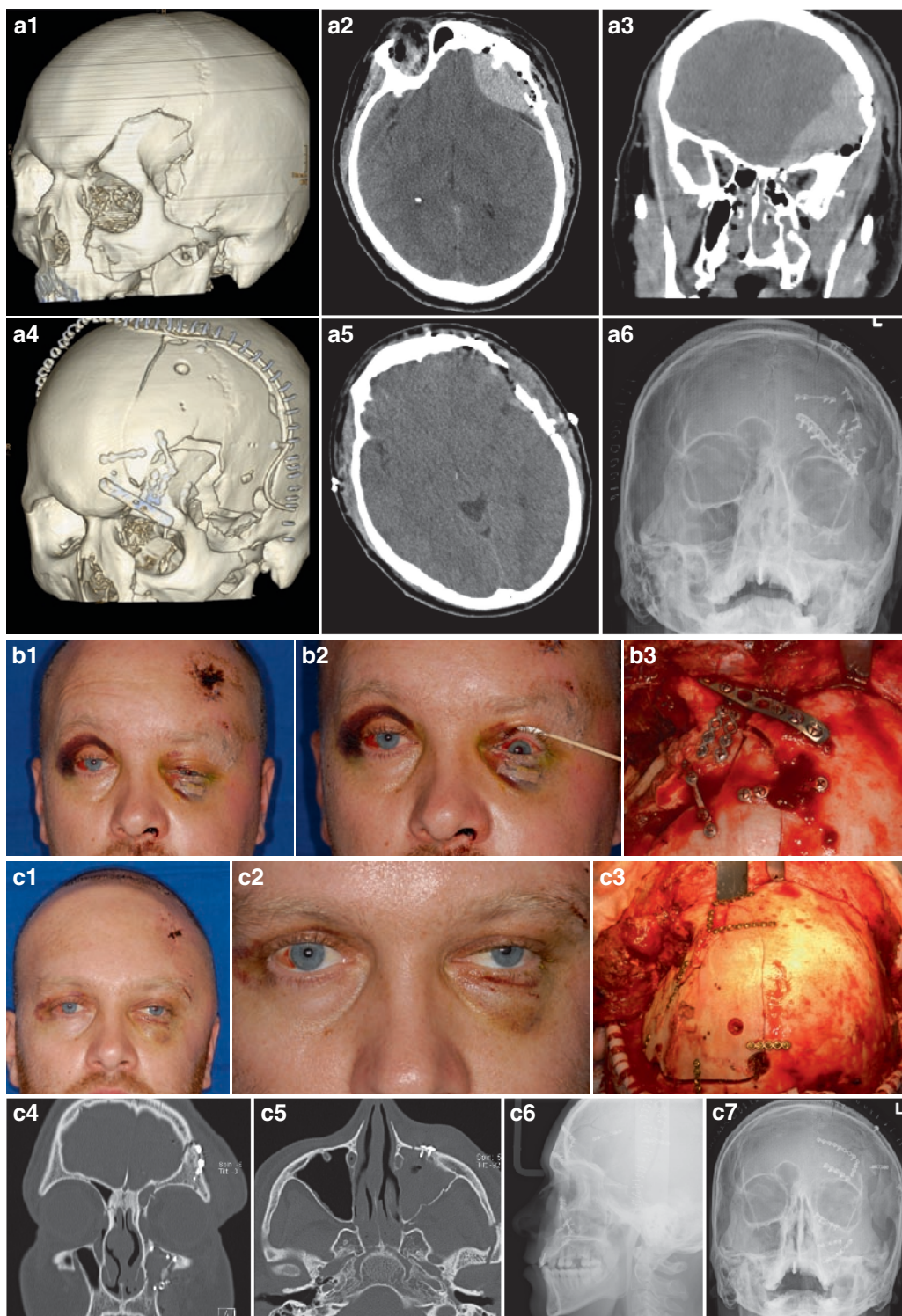


Fig. 12.1 (a) Craniofacial and anterior skull base fractures (COF) with left epidural hematoma. (b) Malposition of the left zygomatico-orbital complex after initial emergency treatment. The cranio-temporal fractures were fixed without simultaneous

midface reconstruction. Low-lying globe as a result of an anatomically incorrect position of the lateral cranio-temporal compartment. (c) Second stage intervention with correct reduction and stabilization of the left zygomatico-orbital complex

Sequence of reconstruction

- Cranio-caudal reconstruction of the external facial frame, beginning with reduction and osteosynthesis of the zygomatic complex and the lateral facial struts and connection to the reduced maxilla (Gruss and McKinnon 1986; Gruss and Phillips 1989; Gruss et al. 1990; Manson 1998, 1999)
- Reconstruction of the internal facial frame, including reconstruction of the periorbital osseous structures and stabilization of the midface struts (Prein 1998; Manson et al. 1999)

3. Reconstruction of the maxillo-mandibular complex and occlusal restitution

Reconstruction in maxillo-mandibular fractures starts with restoring normal occlusion. The occlusion is presumed to be a reliable point of reference for reconstructing the vertical and sagittal lower midface projection, which is lost by telescoping or displacement of the midface structures (Kelly et al. 1990; Wolfe and Berkowitz 1989; Krafft et al. 1991; Sailer and Gratz 1991; Seidl et al. 1998).

The fractured lower midface complex is brought into correct maxillo-mandibular relation by mobilization and reduction, and subsequently using intermaxillary fixation, in centric occlusion.

Sequence of reconstruction (Haerle et al. 1999)

- Reduction of the lower maxilla to restore centric occlusion
- Maxillo-mandibular fixation in centric occlusion. The reconstruction of correct occlusion is fundamental for all further surgical steps.
- Coronal or transfacial approaches for midface reconstruction.

In case of a simultaneous sagittal fracture of the maxilla, the fracture is reduced and stabilized by a

horizontal plate osteosynthesis in the subapertural and palatal regions, resulting in a stable reconstruction of the above (Manson et al. 1990; Haerle et al. 1999). Correct reconstruction of the lower midface level and accurate positioning of the upper facial level prevents a dorso-caudal malposition of the midface. The dislocated central midface can be placed exactly between the lower and upper horizontal struts. In panfacial fractures (PFs), the fractured lower jaw has to be stabilized first.

12.1.2 Surgical Approaches (Manson 1998; Gruss 1990; Prein 1998)

- Soft-tissue approaches

Exposition and treatment of craniofacial fractures is based on thorough planning and necessitates various facial incisions. As a rule the facial frame is exposed using a combination of a coronal incision, transfacial incisions and intraoral incisions (Gruss 1992; Lange et al. 1995; Prein and Luscher 1998; Häerle et al. 1999) (Fig. 12.2).

12.1.3 Reconstruction of Anterior Skull Base and Frontofacial Compartment (Manson 1998; Donald 1994, 1998a; Baker et al. 1999; Gruss et al. 1999; Prein 1998)

- Extradural exposition of the anterior skull base

If there is no intradural pathology which requires treatment, fractures of the frontal sinus, anterior skull base and orbital roofs are treated using the transfrontal-extradural approach.

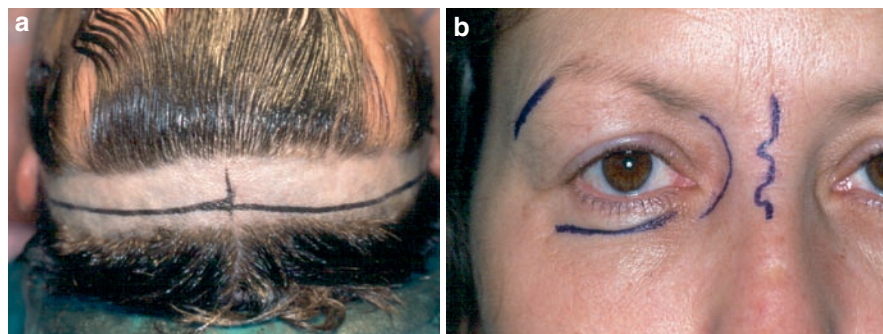


Fig. 12.2 Coronal (a) and transfacial approaches (b) to the upper and middle midface region (medio-nasal/fronto-orbital/medio-palpebral/latero-orbital)

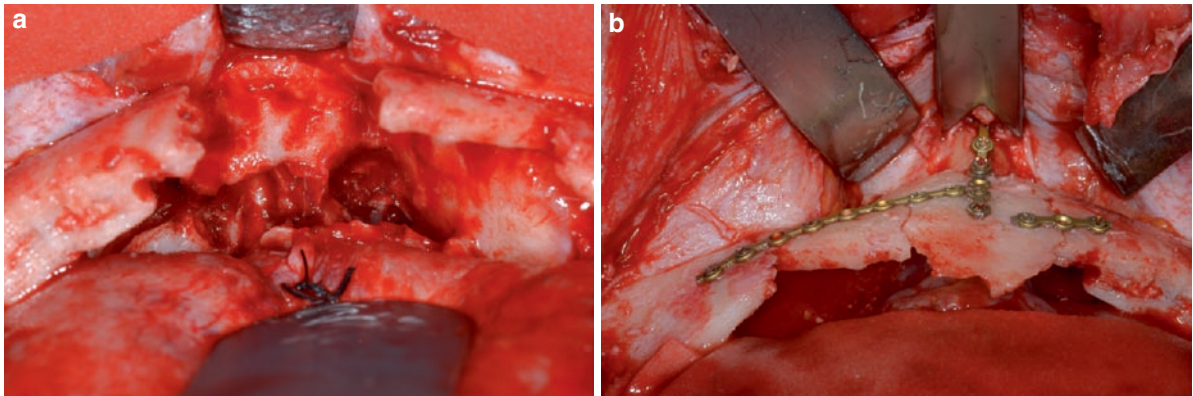


Fig. 12.3 Surgical treatment of anterior skull base fractures. (a) Craniotomy, selective osteotomy of the median fronto-facial band, cranialization of the frontal sinus and debridement of the fractured ethmoidal cells. (b) Complete frontofacial reconstruction

Principle: Exposure of the craniofacial fracture region, temporary removal of comminuted and dislocated fronto-orbital bone fragments and selective craniotomy. Detachment of the dura from the anterior skull base as far as the margin of the sphenoid wing. If necessary, removal of the comminuted posterior wall of the frontal sinus and the ethmoidal roof.

Strongly comminuted ethmoidal cells are debrided, together with the mucous membranes, and drained into the nasal sinus. If fractures are present without significant comminution, treatment can be carried out without performing an ethmoidectomy using a cranial or, alternatively, endonasal or frontoorbital approach. This is also possible when the posterior ethmoid and sphenoid sinus are involved (Imhof 2000; Ernst et al. 2004) (see Chap. 9.3.3.1) (Fig. 12.3).

- Intradural exposition of the anterior skull base.

Principle: The dura is opened and potential contusional hematomas removed. After carefully raising the frontal cerebral lobe, this approach permits a tangential view over the entire frontal skull base whilst selectively protecting the olfactory fibers.

Prior to reduction of the skull base and craniofacial fractures, prolapsed brain tissue has to be released from the fracture lines and repositioned. Trapped, necrotic brain tissue is cut and removed (Schmidek and Sweet 1988).

Subsequently, the dura is loosely adapted and the fronto-facial region is reconstructed. Final intradural treatment of the basal dural lacerations follows frontofacial reconstruction and treatment of the osseous base (see Chap. 9.3.3.2).

- Frontofacial reconstruction

Principle: Following neurosurgical treatment of the cerebral injuries the frontofacial (cranio-frontal) region is subsequently reconstructed. Correct anatomical reconstruction of the cranio-frontal compartment and the zygomatic complex as a guideline for the reconstruction of the facial skeleton in the sagittal and transverse dimensions using miniplate osteosynthesis.

This compartment is an important landmark for correct anatomical reconstruction of the midface skeleton. The reconstruction process follows the bridge building principle, in such a way that the defect is reconstructed starting from the temporal borders and step by step using the available fragments and, if necessary, bone grafts stabilized with 1.5/1.3-mm miniplates. The smaller fragments are fixated using microplates.

This facilitates the subsequent reconstruction of the orbito-zygomatic complex and the correct reduction and osteosynthesis of the midface complex (Ioannides et al. 1988) (Fig. 12.4).

- Skull base treatment

Principle: Subsequent reconstruction of the frontofacial region and treatment of dural injuries; definitive treatment of the skull base; pericranial flap coverage of the revised anterior skull base.

Definitive skull base treatment follows reconstruction of the frontofacial region and neurosurgical treatment of the cerebral and dural injuries. Using the cranial approach, the debrided ethmoid cells are covered with a muscle or fascial patch and fibrin glue, following previous reduction of the orbital roof and anterior skull base

fragments (Imhof 2000; Ernst et al. 2004) (also see Chap. 9.3.3.1).

- Definitive treatment of the anterior skull base has to follow reduction and fixation of the upper midface

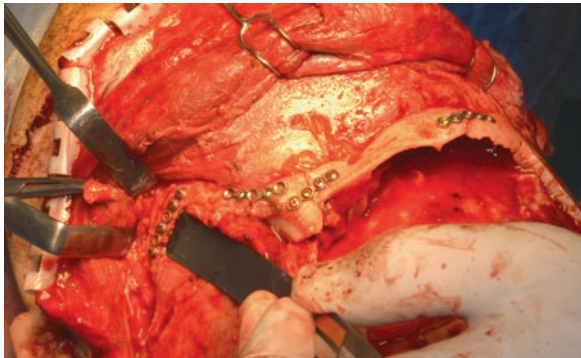


Fig. 12.4 Reconstruction of the frontofacial bandeau following skull base exposure and simultaneous reconstruction of the zygomatico-orbital structures and zygomatic arch

and the fronto-facial bandeau (Joss et al. 2001) (Fig. 12.5)

In the case of extensive skull base defects or missing fragments, additional bone grafts are used (Merville 1985; Rowe and Williams 1985). In grossly comminuted fractures of the anterior and posterior walls of the frontal sinus, reconstruction of the anterior wall is combined with cranialization of the sinus.

The nasofrontal ducts are occluded by dissecting the mucosa, which is displaced caudally and invaginated. The ducts are covered with hemostyptic gauze, fibrin glue and a muscle patch. Thereby the frontal sinus is integrated into the cranial cavity.

Subsequently, the pericranial flap is placed over the reconstructed fronto-facial bandeau and spread on the anterior skull base. During reconstruction of the frontal bone, a slot is left to avoid any compression of the pericranial flap. Tack-up sutures are placed for hemorrhage prophylaxis (Fig. 12.6).

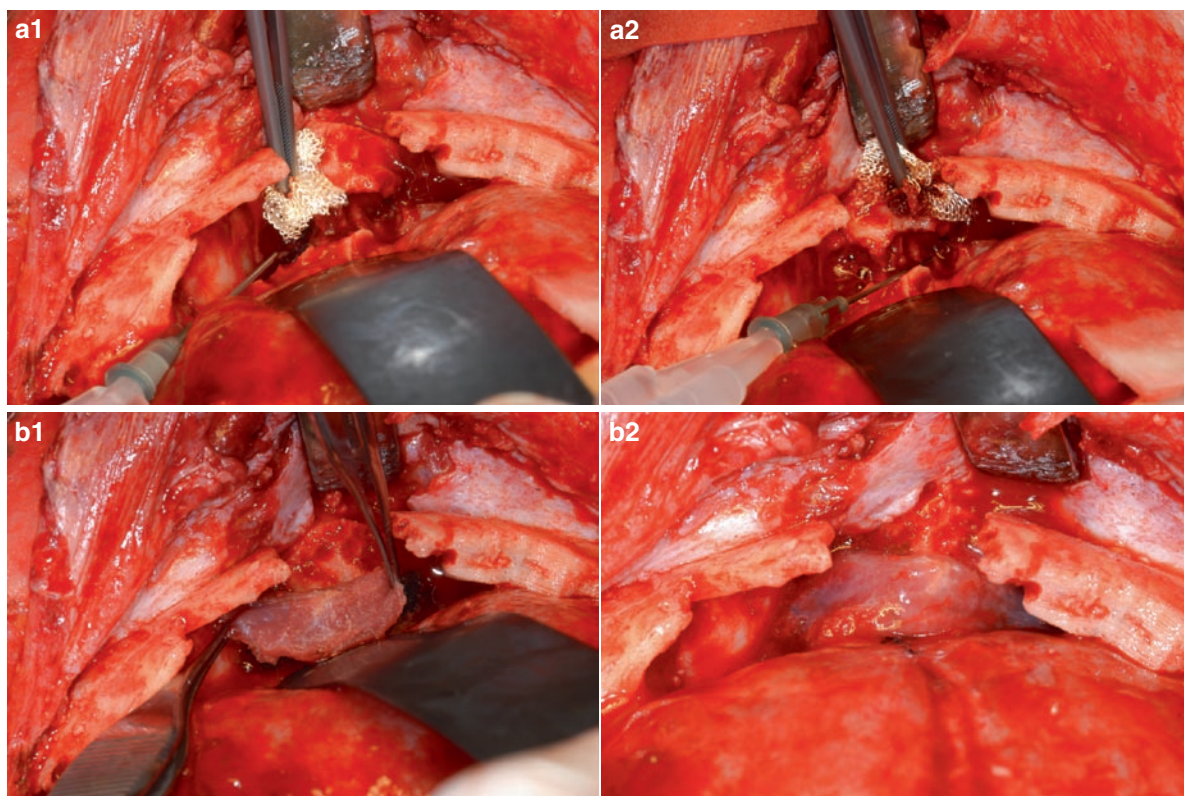


Fig. 12.5 Treatment of a skull base fracture. Occlusion of the frontal sinus floor (nasofrontal duct) and ethmoid roof defects with hemostyptic gauze, fibrin glue (a) and a muscle patch (b)

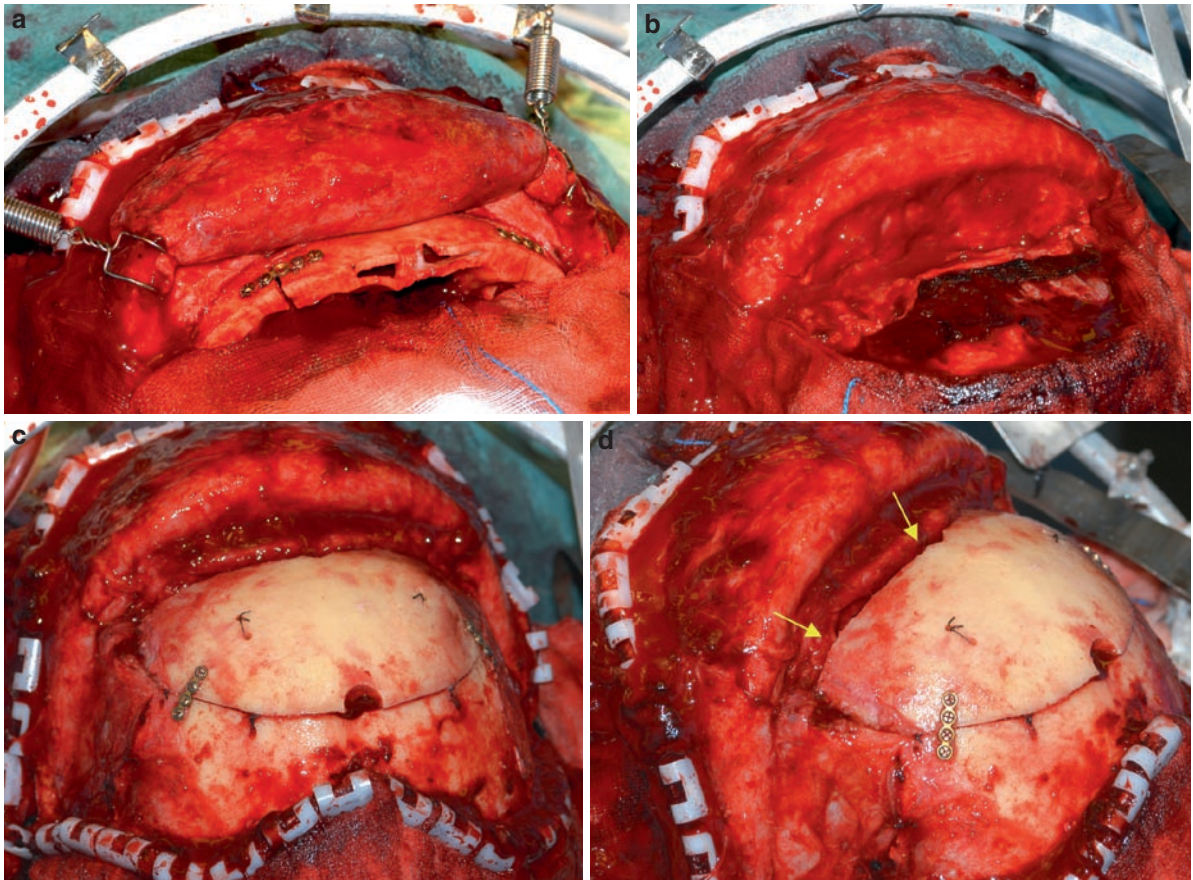


Fig. 12.6 Skull base reconstruction following craniotomy and median fronto-facial osteotomy. (a) Reintegrated and stabilized frontofacial segment. (b) Following skull base treatment, the

vascularized pericranium flap is placed over the frontal skull base. (c, d) Reintegrated calvarian cap with slot for the pericranial flap (arrows), tack-up sutures

- Reconstruction of the fronto-glabellar region

Principle: Reintegration of the calvarial fragments and the frontal bone flap. Osteosynthesis with mini- or microplates. Scalp closure with subgaleatic drainage.

Following skull base treatment, application of tack-up sutures and meticulous hemostasis, the dura is covered with a thin layer of hemostyptic gauze (Tabo-tamp) and reconstruction of the frontal region with reintegration of the calvarian fragments is initiated.

It is important to note that even bone fragments from open fractures can be replaced after thorough cleansing and placement in an antibiotic solution. The bone usually heals without infection.

Depending on the extent of craniofacial fragmentation, integration and fixation of the multiple fragments may be time-consuming. The smaller fragments are fixed using microplates (Sullivan and Manson 1998) (Fig. 12.7–12.11).

- Primary contour adjustments

Particular attention is paid to an aesthetically acceptable reconstruction in the visible section of the forehead, so avoiding secondary corrections as far as possible. Craniotomy holes and gaps are therefore filled with bone dust or milled bone grafts or, alternatively, covered with titanium meshes to prevent postoperative contour irregularities (Kuttenberger and Hardt 2001).

Small, osseous defects in the frontal region are covered with available bony fragments and, if necessary, with bone grafts from the inner or outer table, which are stabilized with microplates. In the case of larger defects, split-skull-grafts from the inner table can be used (Figs. 12.12 and 12.13).

- Fragment healing

In 92%, complete healing of the cranial bone fragments was observed during removal of the osteosynthesis



Fig. 12.7 Stepwise reconstruction of the fronto-glabellar region, beginning with the frontal bandeau and subsequent reintegration of the previously stabilized calvarian fragments

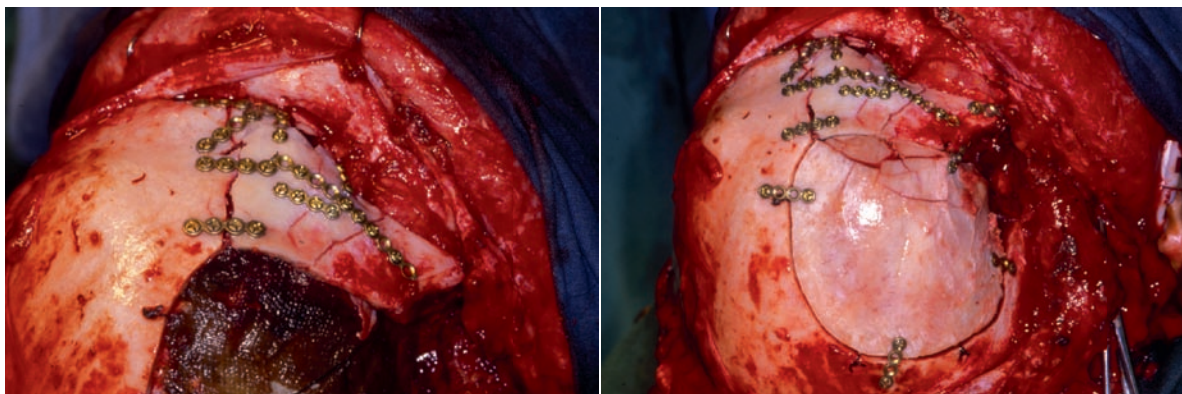


Fig. 12.8 Successive reconstruction and osteosynthesis using 1.5-mm miniplates after complex cranio-orbito zygomatico-temporal fracture with skull base injuries

plates (10–12 months postoperatively). Usually there is complete interfragmentary ossification around the craniotomy line; however, a slight fibro-osseous gap may remain. Histologically, revitalization and preservation of the typical bony architecture of the fragments could be demonstrated.

In 70%, there was no clinically relevant bone resorption; in 30%, there were remaining gaps indicating insufficient bone formation (Hardt et al. 1994) (Fig. 12.14).

12.1.4 Midface Reconstruction

(Yaremchuk et al. 1992; Manson et al. 1995; Manson 1998; Prein 1998; Greenberg and Prein 2002; Gruss et al. 1992, 1999)

Principle: Reduction and stabilization of the midface from cranial to caudal, particularly of the zygomatico-orbital and naso-ethmoidal complex. Restitution of the normal intercanthal distance. Osteosynthesis of the reduced lower midface.

- Reconstruction of the external frame

In reconstructing the midface, primarily the external facial frame is reconstructed, starting from cranial (Gruss and McKinnon 1986; Joos et al. 1989; Prein and Hammer 1988; Gruss 1990; Gruss et al. 1989, 1992, 1999; Prein 1998; Mathog 1992; Manson 1998).

Reconstruction of the external facial frame begins with reduction and osteosynthesis of the zygomatico-orbital complex (Gruss et al. 1990) at the dorsal roots of the zygomatic arch, continuing to the lateral orbit (zygomatico-frontal suture) and to the infraorbital margin (Wolfe and Berkowitz 1989; Krafft et al. 1991; Sailer and Gratz 1991; Jensen et al. 1992). Caudally this is followed by osteosynthesis of the lateral zygomatico-maxillary buttress, which connects the midface to the maxilla (Fig. 12.15).

- Reconstruction of the internal frame

Following restoration of the external transverse and sagittal midface projection, one continues with the reconstruction of the internal frame, beginning with the naso-orbito-ethmoidal (NOE) complex.

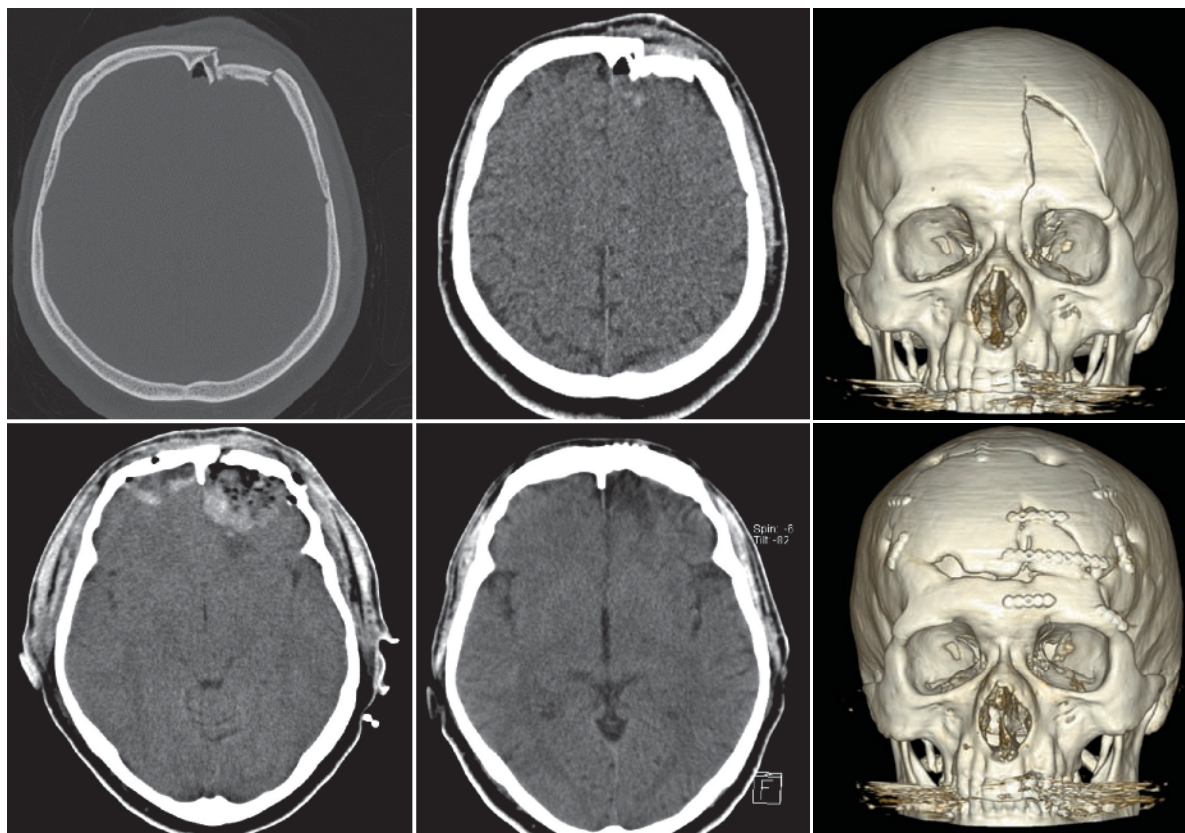


Fig. 12.9 Cranio-orbital impression fracture (COF). The initial CT (**a–c**) shows an impression fracture of the frontal bone with subdural air inclusion (**a**), small contusion hemorrhage in the frontal lobe (**b**) and downward displacement of the large fronto-orbital bone fragment into the medial part of the orbit. Postoperative CTs (**d–f**) show secondary hemorrhage in the fron-

tal lobe in the early postoperative CT (**d**) and postcontusional defects in the pole of the frontal lobe after 3 months (**e**). Anatomic reconstruction of the orbit and good result of interfragmentary plate fixation of the frontal bone fracture (**f**). Frontal craniotomy was performed to repair the dura

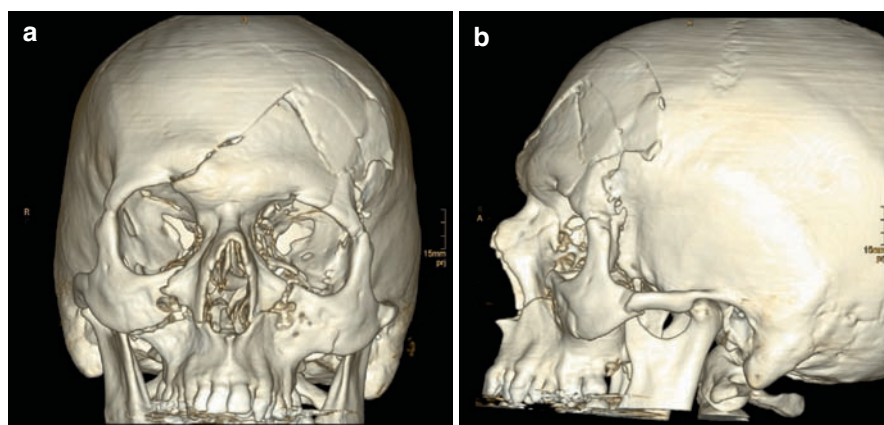


Fig. 12.10 Reconstruction frontal area with several miniplate osteosynthesis (**c, d**) after complex impression fracture of the frontal bone associated with Le Fort-II fracture on the *right side* and zygomatico-orbito-naso-maxillary fracture on the *left side* (**a, b**)

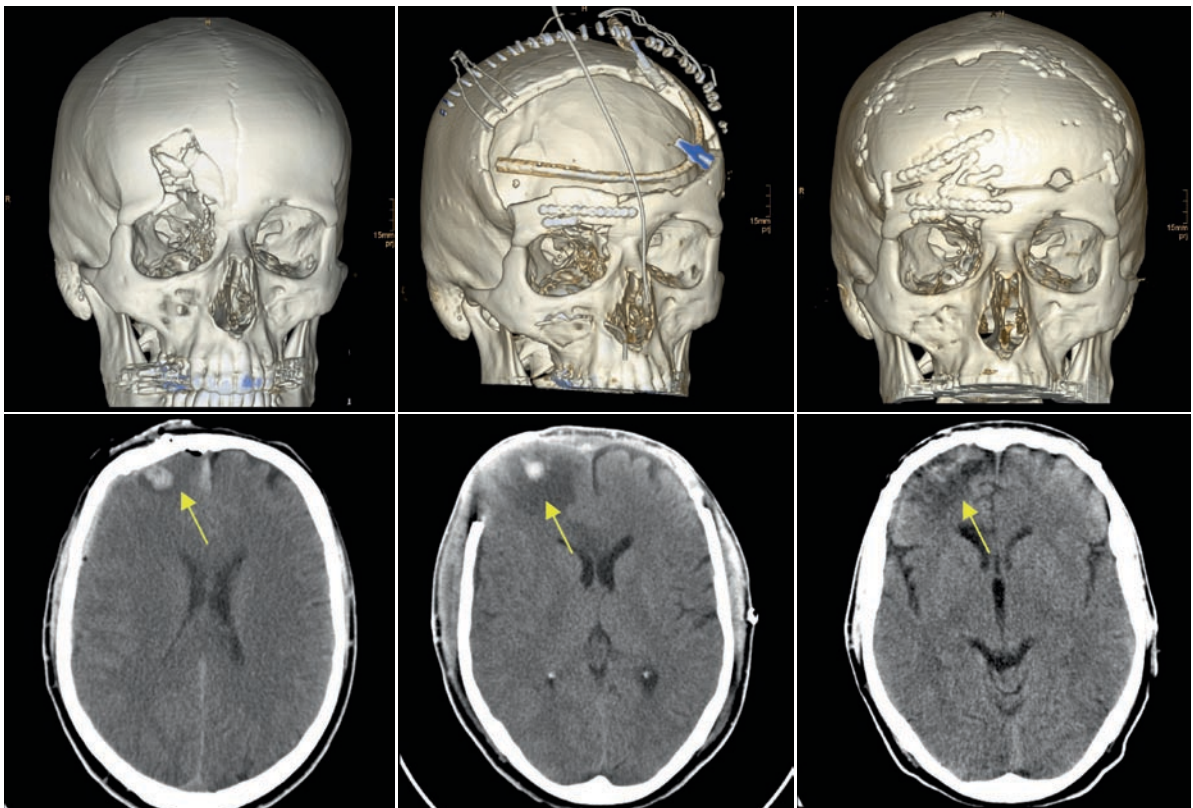
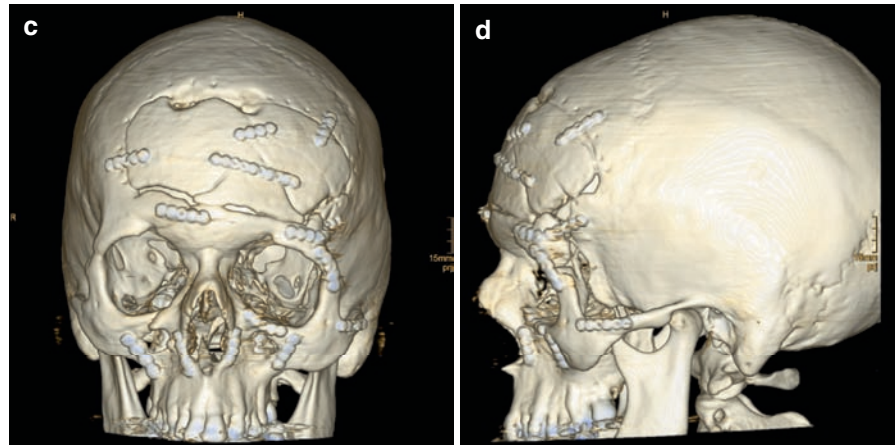
Fig. 12.10 (continued)

Fig. 12.11 Frontoorbital impression fracture (CFF fracture) with brain injury. Comminution of the medial part of the orbital roof (a) and brain swelling and small contusional hemorrhage in the initial CT examination (d) (arrow). First reconstructive step with restitution of the orbital contours and plate fixation and

replantation of the frontal bone flap (b). Craniectomy of the frontal bone is performed to avoid intracranial hypertension (arrow) (e). After relief of brain swelling, replantation of the frontal bone flap is performed, giving a good result (c) with a small post contusional defect in the right frontal lobe (f) (arrow)

After reduction, the NOE complex is fixed in the frontoglabellar region and then connected to the external frame. When connecting the central midface to the external frame, reduction of the upper midface

fracture is completed by reconstructing the periorbital ring.

Particular care must be taken when reattaching the medial canthal ligaments using microplates or transnasal

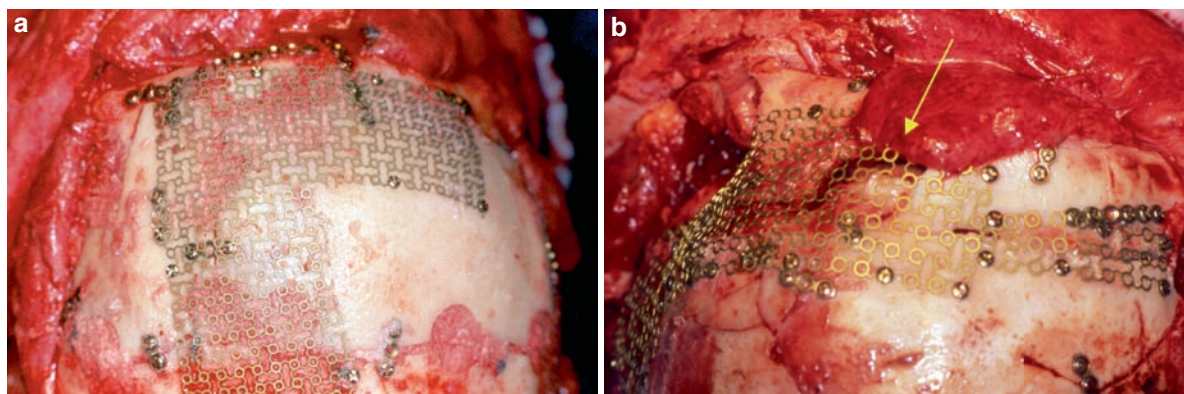


Fig. 12.12 (a) Frontal contour adjustments with bone dust and 0.3-mm titanium micromesh. (b) A 0.3-mm titanium mesh is used for zygomatico-orbital contour restoration (note the

fronto-lateral pericranium flap (*arrow*) which is introduced through the fracture gap

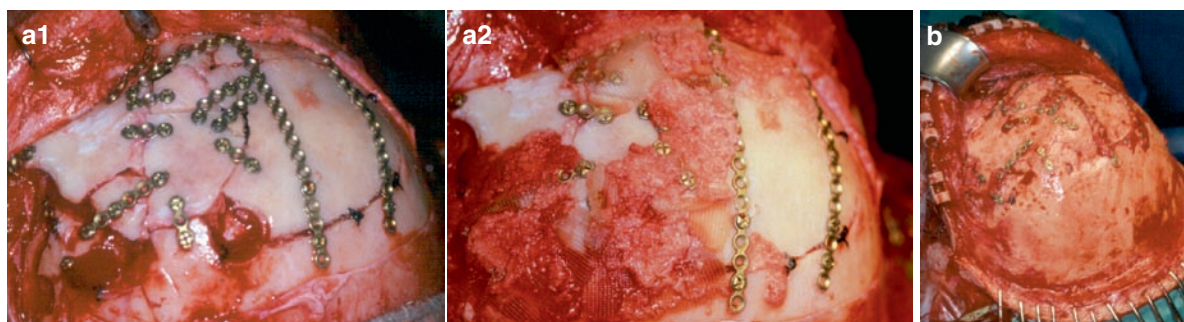


Fig. 12.13 Contour adjustments with bone-dust following reconstruction of a depressed fronto-temporal skull base fracture. (a) Contour adjustments with bone dust following osteosynthesis. (b) Status 1.5 years following reconstruction

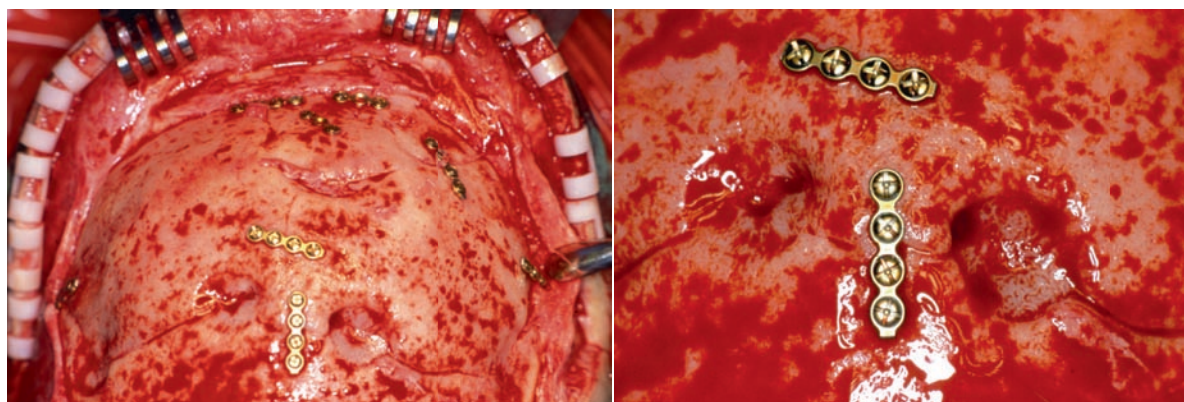


Fig. 12.14 Resorption-depleted fragment healing 1.2 years following fronto-cranial reconstruction (CCMF). Incomplete remodeling with contour impairment in the region of the craniotomy holes

ligatures, to avoid an unacceptable telecanthus (Markowitz et al. 1991; Manson et al. 1999) (see Chap. 12.2.1).

Subsequently, one continues caudally with osteosynthesis of the medial midface struts (infraorbito-

naso-maxillary) and at the Le Fort-I level (piriform aperture/crista zygomatico-alveolaris). If necessary, bone grafts are used (Gruss and McKinnon 1986; Gruss and Phillips 1992).

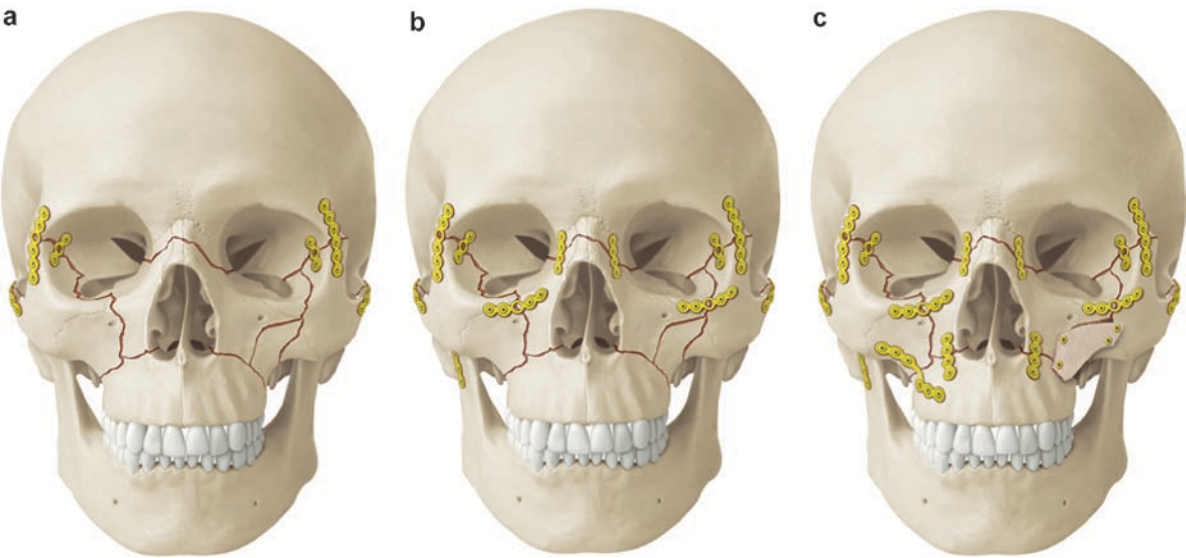


Fig. 12.15 Midface reconstruction, with initial positioning and stabilization of the zygomatico-orbital complex in the cranial and zygomatic arch region (a). Subsequent precise anatomical reconstruction of the central midface and fixation to the recon-

structed external frame and to the fronto-glabellar compartment. Bone grafts are used in cases of buttress structural deficits (b). Finally stabilization of the reduced lower maxillary fragments (medial and lateral bone struts) (c) (mod. a. Prein 1998)

12.1.5 Own Procedure: Statistics

In 75%, craniofacial reconstruction was performed using miniplate osteosynthesis only. In 10%, additional bone grafts were used, and in 12%, titanium meshes. In 3%, a combination of miniplates, bone grafts and titanium meshes was necessary (Neidhardt 2002).

(Yaremchuk et al. 1992; Manson et al. 1995; Manson 1998; Donald 1998a; Prein 1998; Greenberg and Prein 2002; Gruss et al. 1992,1999).

Craniofacial reconstruction techniques in craniofacial fractures (n = 197) (Neidhardt 2002)

Reconstruction with miniplates only	75%
Reconstruction with miniplates and titanium mesh	12%
Reconstruction with miniplates and bone grafts	10%
Reconstruction with miniplates, bone grafts and titanium mesh	3%

12.2 NOE Fractures

Fractures of the central midfacial compartment involve the NOE complex and, optionally, the skull base (Gruss 1982, 1986; Probst and Tomaschett 1990; Prein 1998; Oeltjen and Hollier 2005).

Dependant on the complexity of the trauma, fractures of the ethmoid always bear the risk of associated

skull base fractures with intracranial injuries, also necessitating a neurosurgical approach (Ayliffe and Ward-Booth 2003).

Principle: Transfacial or transfrontal-subcranial approach, debridement of the fractures, proper treatment of the upper paranasal sinuses and exploration of the skull base, if necessary with dural repair. Reconstruction of the nasal and interorbital structures with microplate-osteosynthesis, paying attention to the intercanthal distance. Reattachment of the medial intercanthal ligaments and reconstruction of the medial orbital walls (Duvall et al. 1981; Merville et al. 1983; Gruss 1986; Markowitz et al. 1991; Leipziger and Manson 1992; Mathog et al. 1995; Ayliffe and Ward-Booth 2003).

12.2.1 NOE-Fractures Without Skull Base Injury (Prein 1998; Donald 1998a, b)

Based on the NOE-classification from Markowitz et al. (1991) and Mathog et al. (1995), reconstruction is performed according to the following steps:

Following reduction, telescoping *type 1-fractures* are stabilized with 1.5/1.3-mm miniplates along the nasofrontal process (Mathog 1992; Vuillemin et al. 1988) (see Fig. 11.21a):

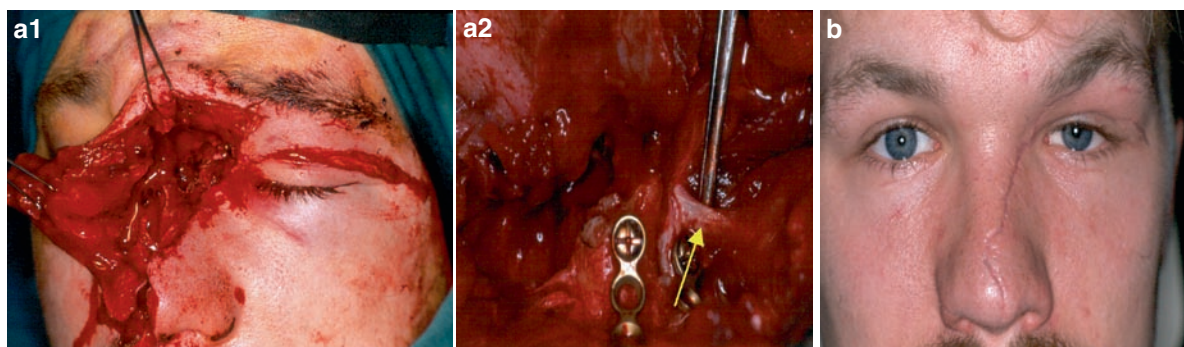


Fig. 12.16 NOE-fracture with canthal ligament type-2 injury (bone-ligament avulsion). (a) Intraoperative site before and after osteosynthesis of the frontal process and before fixation of the

central bone fragment with adherent canthal ligament (*arrow*). (b) Postoperative result

- Superiorly to the fronto-glabellar region
- Inferiorly to the maxilla

In the case of multi-fragmental NOE injuries *type 2 and 3*, the fragments are reduced and temporarily stabilized with wire osteosynthesis so that the entire complex can be definitely fixed to the stable surrounding midface structures — beginning at the medial orbital border — superiorly to the cranial complex and caudally to the maxillary complex (Prein 1998) (see Fig. 11.21b, c).

Fractures of the central and dorsal ethmoid can be simultaneously treated using the endonasal approach.

- Canthal ligament fixation

Transnasal wire osteosynthesis stabilizes the NOE complex, restores the normal intercanthal distance and re-establishes the anatomically correct ligament insertion (Laedrach et al. 1999; Maniglia et al. 1999; Haerle et al. 1999; Ward-Booth et al. 2003).

- Type 1 canthal ligament injuries (avulsion of the medial canthal ligament)

The canthal ligament is fixed by a transnasal titanium wire. The wire is secured contralaterally using 1.3-mm microplates or miniscrews (Jackson 1989; Prein 1998; Oeltjen and Hollier 2005; Hammer 2001). Alternatively, a barbed titanium wire can be used to fix the canthal ligament (Hammer 2001). After localizing the ligament, the titanium wire is inserted through it and the hooks grip the ligament. The ligament is then correctly placed; the titanium wire is passed transnasally and fixed to the contralateral medial orbital wall. The point of insertion of the ligament lies posterior and superior to the lacrimal fossa (Hammer 1995; Hammer and Prein 1998; Ward-Booth et al. 2003).

- Type 2 and 3 canthal ligament injuries (bone-ligament avulsion)

The displaced central fragment is reduced, together with the attached medial canthal ligament, using a transnasal wire or, if technically possible, stabilized with microplates (Mathog 1992; Mathog et al. 1995). The transnasal wire is fixed contralaterally using a miniscrew or 1.3-mm miniplate (Hammer 2001) (Fig. 12.16).

In the case of missing central bone fragments, the ligament is fixed by guiding the titanium wire through a hole of a 1.3/1.5-mm miniplate bridging the defect on the affected side. The wire is then directed transnasally towards the contralateral side and anchored to another plate (“toggle-plate”) or screw.

Alternatively, the medial orbital wall can be reconstructed with a bone graft. The ligament insertion is then positioned at the stabilized bone graft using a transnasal wire, which is guided through a hole in the graft. (Markowitz et al. 1991; Ellis 1993; Hammer 1995; Hammer and Prein 1998; Oeltjen and Hollier 2005; Stewart 2005) (Figs. 12.17 and 12.18).

12.2.2 NOE Fractures with Skull Base Injury (Prein 1998; Donald 1998a, b)

As a rule, injuries concerning the NOE complex and involving the skull base are complex fractures, necessitating a structured revision. Depending on the extent of injury and accompanying midface fractures, there is a surgical indication for a transfacial subbasal or transfrontal extradural procedure to treat the injuries of the NOE complex.

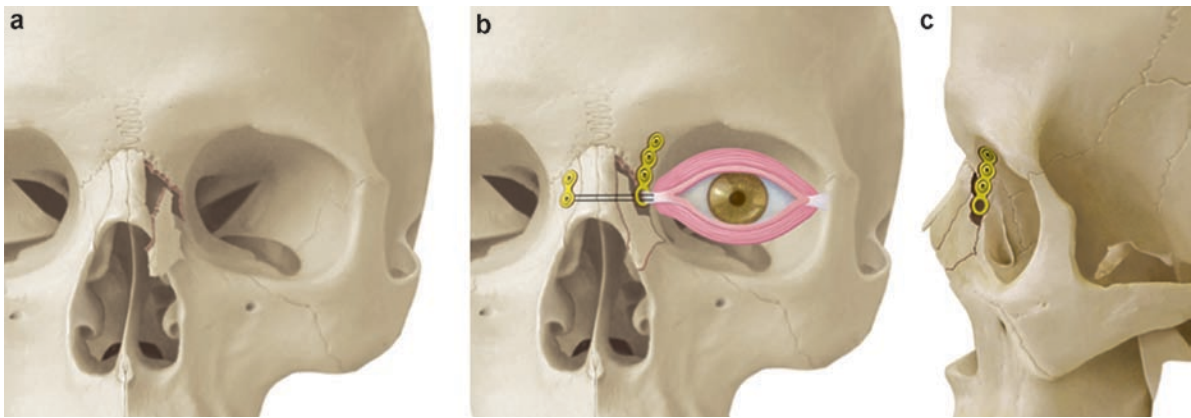
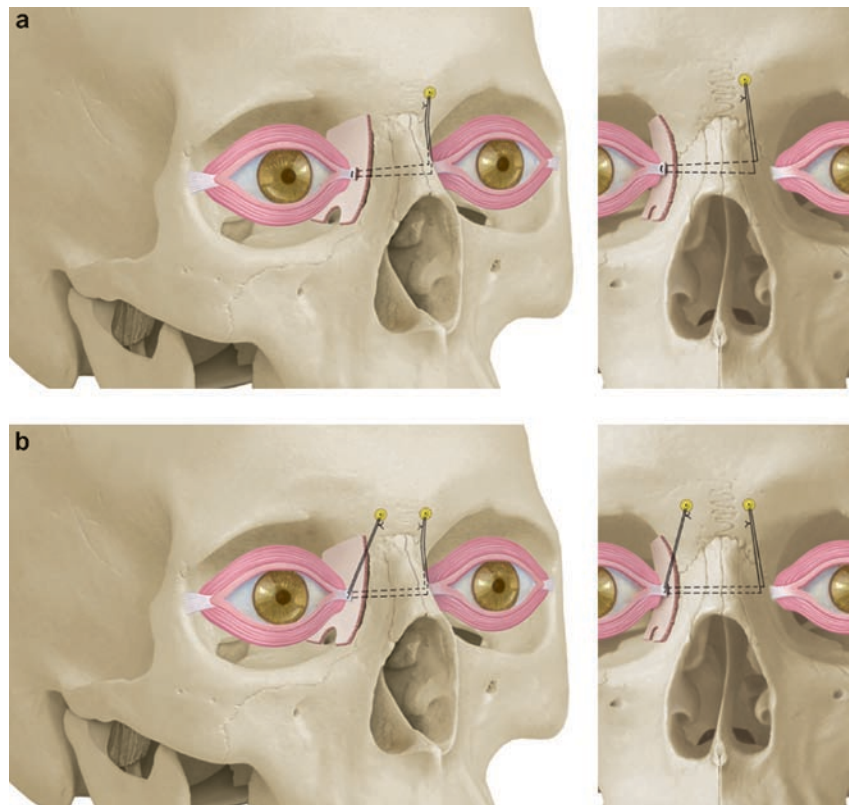


Fig. 12.17 Reconstruction of the medial canthal ligament in type-2 injury with missing bone (a). In case of missing bone (central fragment) at the fracture side the medial canthal ligament is secured by transnasal canthopexy through a positioning

plate. The direct transnasal wire passes the hole of the positioning plate and is contralaterally twisted over a microplate (“toggle-plate”) (b, c) (mod. a. Merville 1985; McMahon et al. 2003)

Fig. 12.18 Reconstruction of the medial canthal ligament in case of missing bone at the fracture side (NOE fracture type 2).

(a) Reconstitution of the medial orbital wall with a bone graft. Securing the medial canthal ligament with a transnasal wire. The wire passes through the bone graft at the fracture site and secures the right medial canthus. (b) Naso-orbital reconstitution with bilateral canthal dystopia in case of unilateral missing bone at one fracture side (NOE fracture type 3). Unilateral repair of the medial orbital wall with a bone graft and bilateral canthopexy. The wires (b) pass through a hole in the bone graft and secure the right medial canthus. The wires (a) fix the left canthal ligament to the central fragment (mod. a. Merville 1985; McMahon et al. 2003)



Reconstructive Sequence (Markowitz et al. 1991; Gruss 1990; Gruss et al. 1985, 1992)

- Reconstruction of the frontonasal strut and the external medial orbital ring either via transfacial naso-orbital or the coronal approach
- Treatment of frontal sinus, skull base, and dura
- Reconstruction of the nasal frame by interfragmentary osteosynthesis
- Reconstruction of the medial orbital wall
- Re-attachment of the medial canthal ligaments
- Reconstruction of the nasal bridge, correcting nasal projection (plate, graft) (bear in mind the frontonasal angle)
- Splinting the nasolacrimal duct

The initial “en bloc” reduction of the naso-ethmoidal complex, often simultaneously reduces the dislocated fractures in the area of the cribriform plate, which resulted from compression of the bony complex.

In case of multifragmentation of the naso-ethmoidal complex with frontal skull base fractures and simultaneous midface fractures, it is advisable to begin with the reconstruction of the naso-fronto-maxillary struts, including the naso-orbital complex, and then continue with the reduction and stabilization of the central midface fractures, before treating the skull base fractures.

12.3 Panfacial Fractures (Markowitz and Manson 1989; Manson 1998; Kelly et al. 1990; Manson et al. 1995, 1999; Marchena and Johnson 2005; McGraw-Wall 2005)

There is no general consensus as to which is the best sequence in treating PFs (Haerle et al. 1999; Hausamen and Schierle 2000), although there is consent that fracture dislocation and extent of comminution are decisive in choosing the surgical procedures (Mathog 1992; Markowitz and Manson 1998; Haerle et al. 1999; McMahon et al. 2003).

Rohrich and Shewmake (1992) advise primary reconstruction of the lower facial level, so enabling correct reconstruction of the dorsal vertical height and lower facial width.

12.3.1 Concept of Reconstruction

The preferred sequence in complex panfacial fractures starts with mandibular reconstruction, including fractures of the temporo-mandibular joints, particularly in case of bilateral condylar neck fractures. As the next step, the fronto-facial and zygomatico-orbital compartments are reconstructed, which are the key for subsequent midfacial reconstruction (Markowitz and Manson 1989; Kelly et al. 1990; Manson et al. 1995, 1999; Haerle et al. 1999; Fonseca 2000; McMahon et al. 2003; Stewart 2005; McGraw-Wall 2005).

Reconstructive sequence (Ward-Booth et al. 2003; McMahon et al. 2003)

- Mandibular reduction and osteosynthesis as well as maxillary reduction and maxillo-mandibular fixation
- Reconstruction and osteosynthesis of the fronto-facial region and the zygomatico-orbital and NOE complex
- Completion of midface reconstruction through osteosynthesis of the external and internal facial frames
- Treatment of the frontal sinus, skull base and dura
- Reconstruction of the nasal frame and orbital cavity (bone grafts if necessary)

1. Treatment of the mandible (lower facial level)

Correct reconstruction of the mandible, including condylar fractures, is considered to be a crucial factor for three-dimensional midface reconstruction (Zide and Kent 1983; Hayward and Scott 1993).

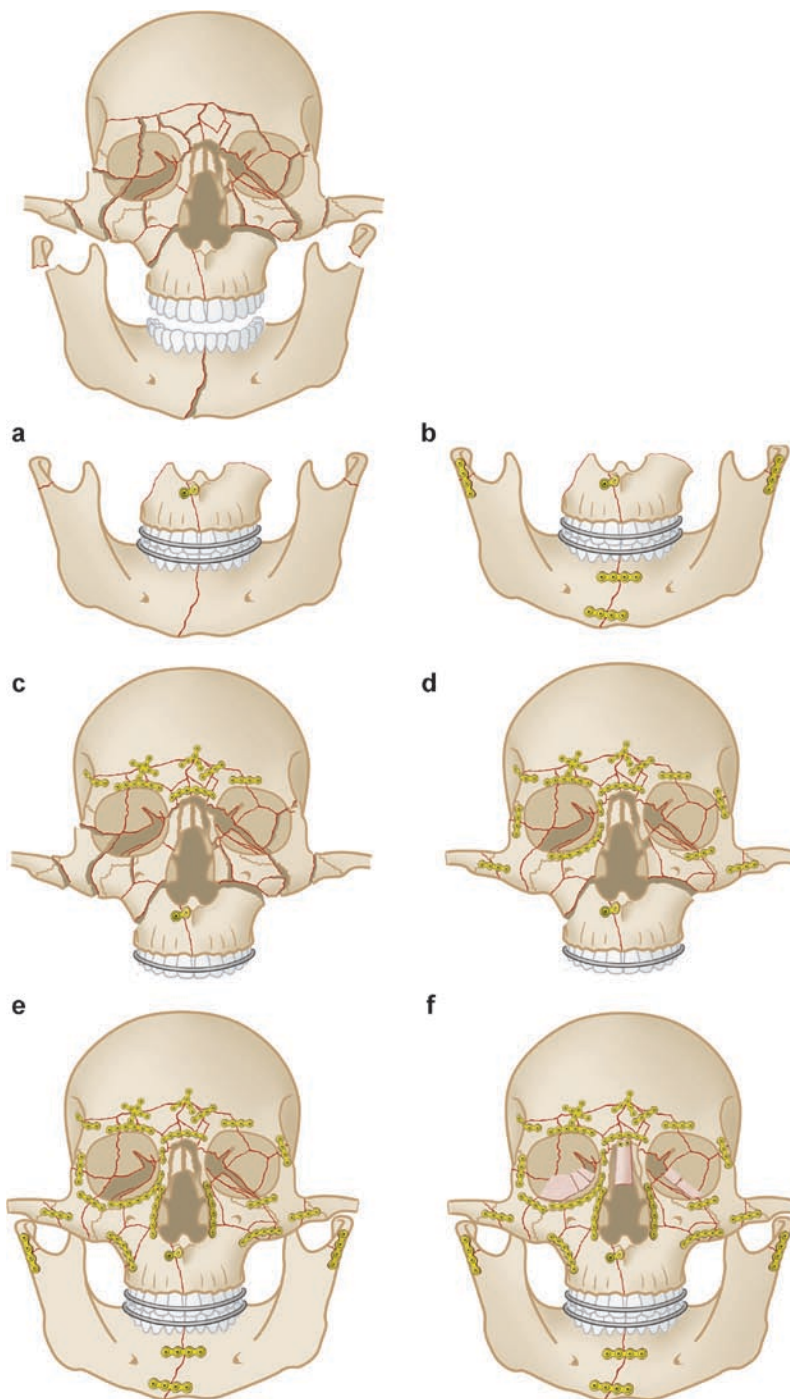
This is the key to avoid loss of anterior-posterior dimensions and posterior vertical lower facial height. The maxilla is then positioned and fixed in centric occlusion in relation to the mandible (Kelly et al. 1990; Manson et al. 1995; Haerle et al. 1999).

2. Treatment of the central and upper midface

Following the precise transverse and sagittal reconstruction of the upper midfacial level (fronto-facial bandeau), the central midface is reconstructed starting with reduction and osteosynthesis of the zygomatic complex and zygomatic arch. Optionally, osteosynthesis of the zygomatico-maxillary buttress (external frame) in the adjusted lower midface can follow, thus

Fig. 12.19 Sequence of operative repair in panfacial injuries (mod. a. Ward-Booth et al. 2003; McMahon et al. 2003; McGraw-wall 2005).

(a) Sagittal fractures of the maxilla and mandibular fractures with flaring of the vertical rami lead to errors with excessive width and deficient anterior projection. Pressure to the gonial angles close any lingual gap in the anterior mandible. A useful guide to the correct reduction is the point at which the anterior fracture just starts to open on its labial or buccal surface. At this point the lingual cortex is acting as a fulcrum. (b) Fixation of the mandibular condyle to prevent excessive width as well as restoring posterior facial height. (c) Reassembling the upper facial subunit (fronto-glabellar) precedes midface repair. (d) Midface repair starts at the least injured part of the orbits (zygomatico-orbital/fronto-naso-orbital) and uses all visible landmarks to establish the correct anterior projection. (e) Buttress reconstruction is completed by miniplate osteosynthesis of the zygomatico-maxillary (external frame) and naso-maxillary (internal frame) vertical buttresses. (f) Orbital bone grafting, nasal bone grafting and medial canthopexy are performed where necessary and prior to inset of a pericranial flap



facilitating the positioning of the central midfacial structures (internal frame).

In the next step, reconstruction of the NOE complex is carried out and, consecutively, the periorbital ring is completed (Haerle et al. 1999; Fonseca 2000; Marchena and Johnson 2005).

3. Treatment of the lower midface

Midface reconstruction is completed with stabilization of the vertical naso-maxillary and zygomatico-maxillary struts. If necessary, a medial canthopexy is finally done and bone grafts are inserted for orbital or nasal reconstruction (Figs. 12.19-12.23).

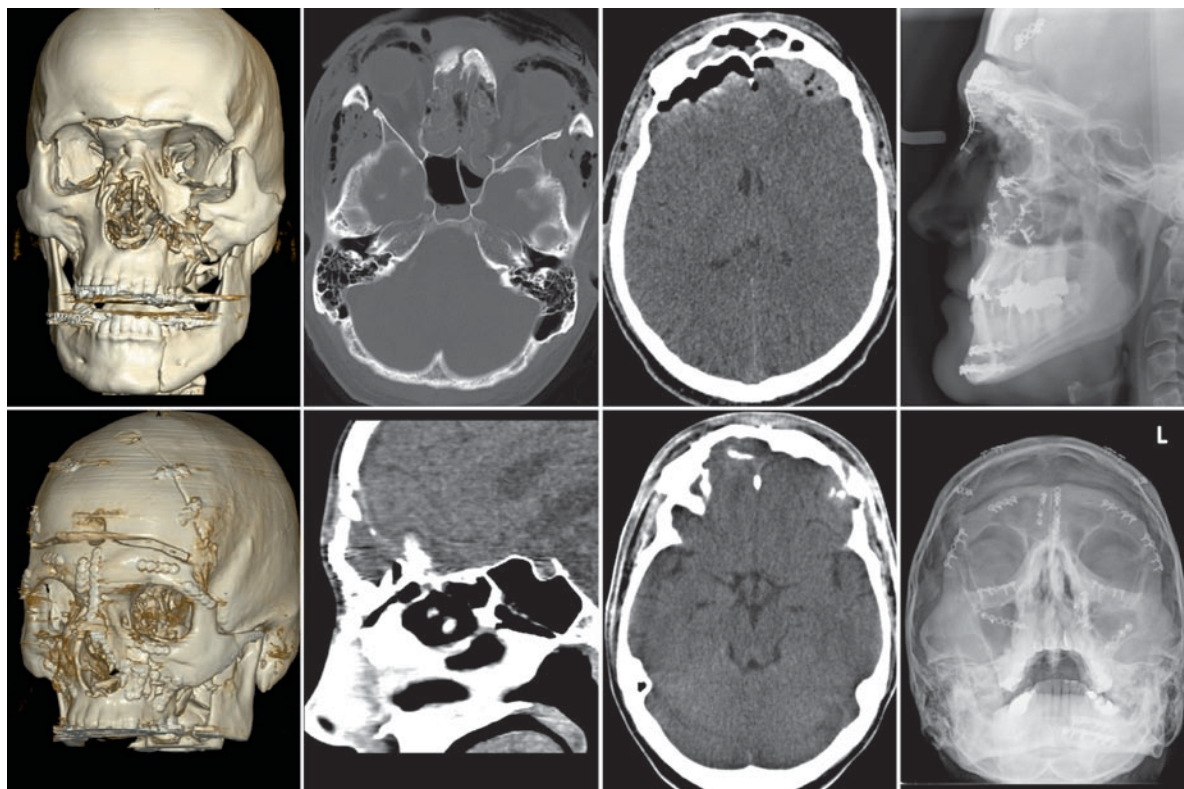


Fig. 12.20 (a–c) Panfacial fracture (frontobasal - NOE-fracture, Le Fort-III, left Le Fort-II, and zygomatico-orbital fracture, mandibular fracture). (d–f) Post-reconstructive result after

reduction and fixation of the midfacial frame with multiple 2.0-mm miniplates. (g–h) Frontal craniotomy and cranialization of the frontal sinus

Fig. 12.21 Panfacial fracture. Internal osteosynthesis with multiple 2.0-mm miniplates after craniotomy, skull base exploration and reconstruction of the frontofacial bandeau





Fig. 12.22 Panfacial fracture (cranio-frontal-fracture, Le Fort-III, left Le Fort-II and sagittal-maxillary fracture, mandibular fracture). (a) Internal osteosynthesis with multiple 2.0-mm mini-

plates after craniotomy, skull base exploration and reconstruction of the frontofacial bandeau. (b) Result 1 year postoperatively

12.4 Zygomatico-Orbito-Cranial Fractures

Zygomatico-orbito-cranial fractures comprise complex lateral midface fractures with additional involvement of the cranial skeleton, the naso-ethmoidal complex and the maxilla. These fractures should always be treated primarily to avoid late functional and aesthetic disturbances (Chuong and Kaban 1986). Orbital reconstruction is preferentially carried out using available bony fragments.

Calvarial bone grafts should be used for primary reconstruction in cases of extensive orbital wall defects (Fonseca and Walker 1991) (Fig. 12.24).

Delayed surgical treatment of these complex fractures is much more complicated and correct reduction of the zygomatico-orbital complex is not always possible. Secondary corrections of zygomatico-orbital malpositions usually require extensive osteotomies and CT-based navigation (Gellrich et al. 2003).

An additional (unilateral) transfrontal craniotomy may be necessary in the following situations:

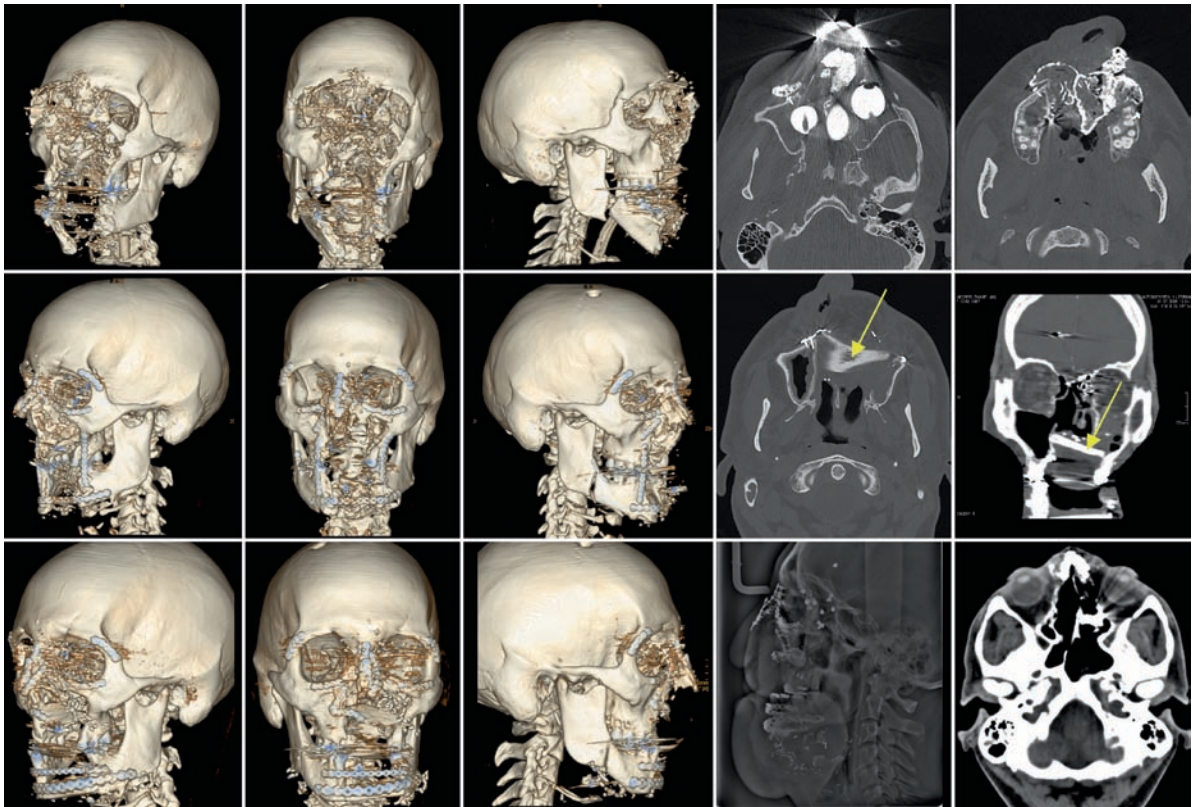


Fig. 12.23 Burst trauma of the midface after severe suicidal gun shot from submental through the midface. *First row*: post-traumatic situation with partial destruction of the central mid-face and loss of the lower central midfacial structures including

the palate. *Middle row*: situation after debridement and reconstruction of the palate with microvascular bone graft (arrow). *Lower row*: after reconstruction of the mandible with 3.0/2.4-mm locking reconstruction plates

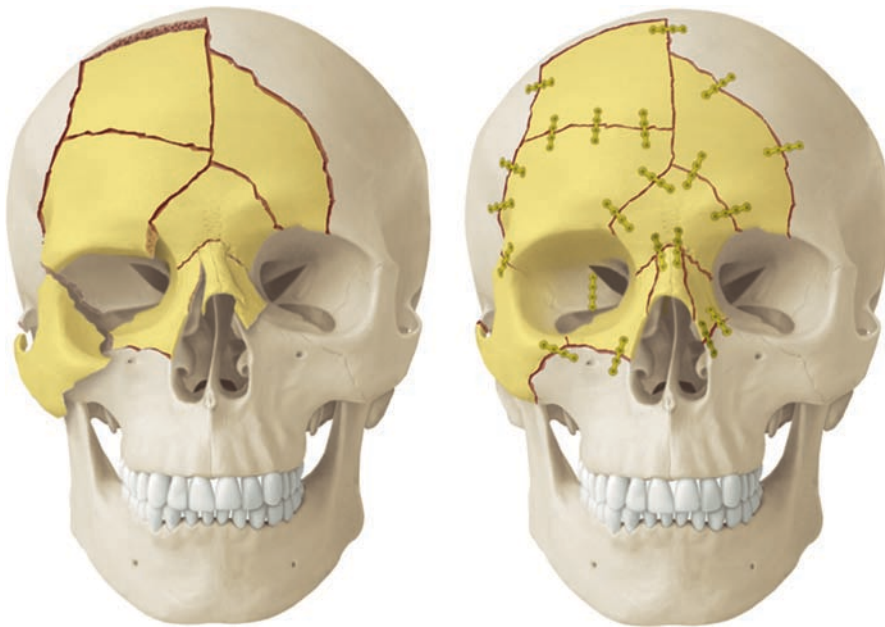


Fig. 12.24 Sequence of stabilization in zygomatico-orbito-cranial fractures, starting with osteosynthesis of the fronto-glabella area, followed by the zygomatico-orbital, and, lastly, the naso-maxillary complex

- Orbito-cranial injuries with
 - Involvement of the orbital roof
 - Orbito-frontal perforations
 - Extensive lateral orbital roof fractures and cerebral or dural injuries
- Isolated, dislocated orbital roof fractures (no fractures of the skull bone)
- Traumatic optical nerve injuries or compression of the optic nerve due to fractures of the sphenoid bone (decompression!) (Fig. 12.25–12.30)

Reconstructive sequence (Samii and Draf 1989; Weerda 1995)

- Transfrontal exposure of the orbito-temporo-cranial region using the coronal approach with pre-auricular extension possibly in combination with transfacial incisions
- Subperiosteal exposure of the zygomatico-orbital and temporal complex by intrafascial preparation with possible lateralization of the temporal muscle
- Fragment elevation and selective osteoplastic temporal craniotomy and treatment of cerebral-dural and orbital injuries
- Reduction and osteosynthesis of the zygomatic arch and the zygomatico-orbital complex; reconstruction of the orbital roof and supraorbital margin as well as the periorbital osseous frame
- Reduction and osteosynthesis of accompanying maxillary/naso-ethmoidal fractures
- Reconstruction of the orbital walls with autogenous bone grafts or resorbable membranes; refixation of the medial/lateral canthal ligaments

12.5 Craniofrontal Fractures (CCF)

In this fracture class, the following fracture subtypes are listed according to the extent of the fracture (Donald 1986, 1998a, b; Duvall et al. 1987; Wolfe and Johnson 1988; Stanley 1989):

- Frontal sinus fractures (isolated)
- Frontal sinus-ethmoidal fractures
- Frontal sinus-orbital-ethmoidal fractures
- Combined fractures

Fractures of the craniofrontal region comprise about 5% of all fractures in the facial region and 2–12% of all cranial fractures (Ioannides et al. 1993).

There may either be isolated or combined fractures of the anterior and posterior walls of the frontal sinus.

As the bone lamellae of the posterior wall are considerably thinner than those of the anterior wall, frontal impact vectors may result in isolated fracture of the posterior wall caused by lateral tension during skull deformation (Hybels 1977).

12.5.1 Concept of Reconstruction

The reconstructive concept is orientated according to fracture extent of the ventral and dorsal frontal sinus walls (types I–III/Godbersen and Kügelgen 1998) as well as the condition and involvement of the ethmoidal infundibulum and the skull base (Levine et al. 1986; Luce 1987; Stanley 1989; Godbersen and Kügelgen 1998; Mathog 1992; Baker et al. 2003).

- Type-I fractures (ventral sinus wall fractures): no concomitant fractures exist. In such cases, no ethmoidal cell debridement is necessary.
- Type-II fractures (ventral and dorsal sinus wall fractures): the dorsal wall of the frontal sinus and the ethmoid region must always be examined to exclude dural injuries. In case of doubt, liquor-marking with fluorescein should be undertaken.
- Type-III fractures are easily recognized, either by rhinoliquorrhea or cerebral prolapse with evidence of dural injury. The more extended the fractures (orbit and midface fractures), the higher the probability of dural injury (Godbersen and Kügelgen 1998).

The principle aim is to reconstruct the frontal sinus (Ehrenfeld et al. 1996; Ernst et al. 2004). Cranialization of the frontal sinus is indicated in comminuted fractures of the posterior sinus wall.

Reconstructive sequence (Baker et al. 2003)

- Transfrontal or transfacial exposure of the frontal region
- Fragment elevation and treatment of cerebral and dural injuries (cranialization of the frontal sinus, if necessary)
- Reconstruction of the anterior wall of the frontal sinus using local bone or autogenous bone grafts (Donald 1986; Ellis 1993). Reconstruction of the anterior wall of the frontal sinus with titanium mesh in case of multiple fractures

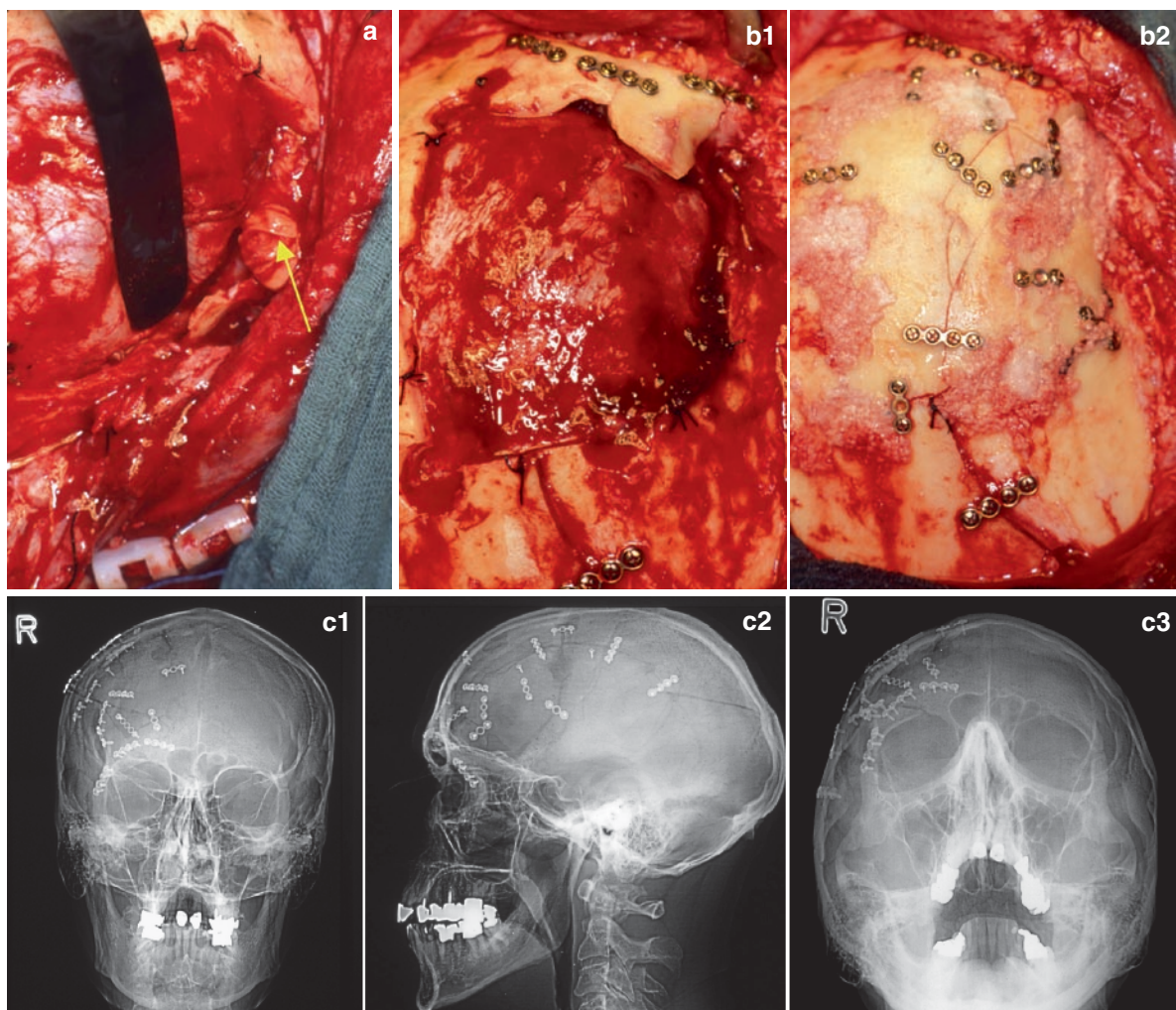
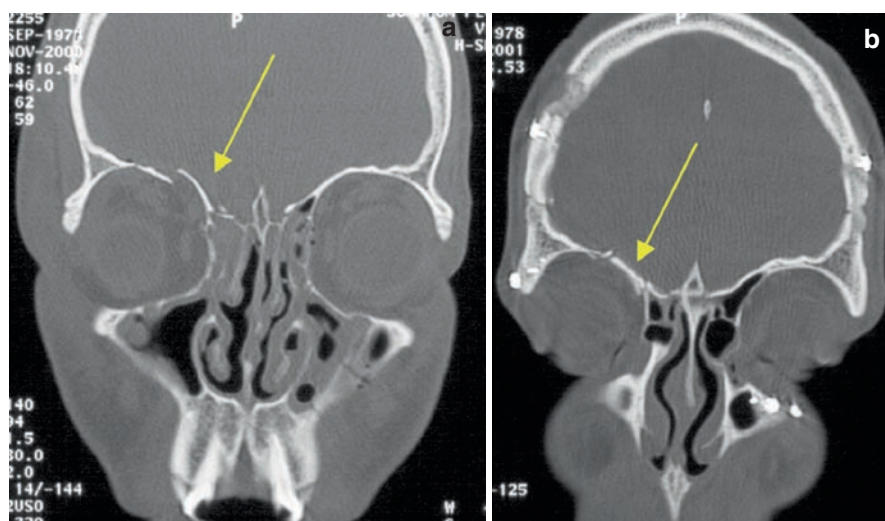


Fig. 12.25 Dislocated cranio-orbital fracture with anterior skull base fracture (orbital roof). (a) Orbital roof fracture and prolapse of the orbital fatty tissues (arrow). (b) Sequential reconstruction of the frontal region. (c) Postoperative X-ray control

Fig. 12.26 Cranio-orbito-maxillary fracture (CCMF). (a) Preoperative CT scan showing displaced fracture of the right orbital roof and ethmoid (arrow). (b) Postoperative radiograph demonstrating exact reduction of the orbital roof fragments (arrow) after craniotomy



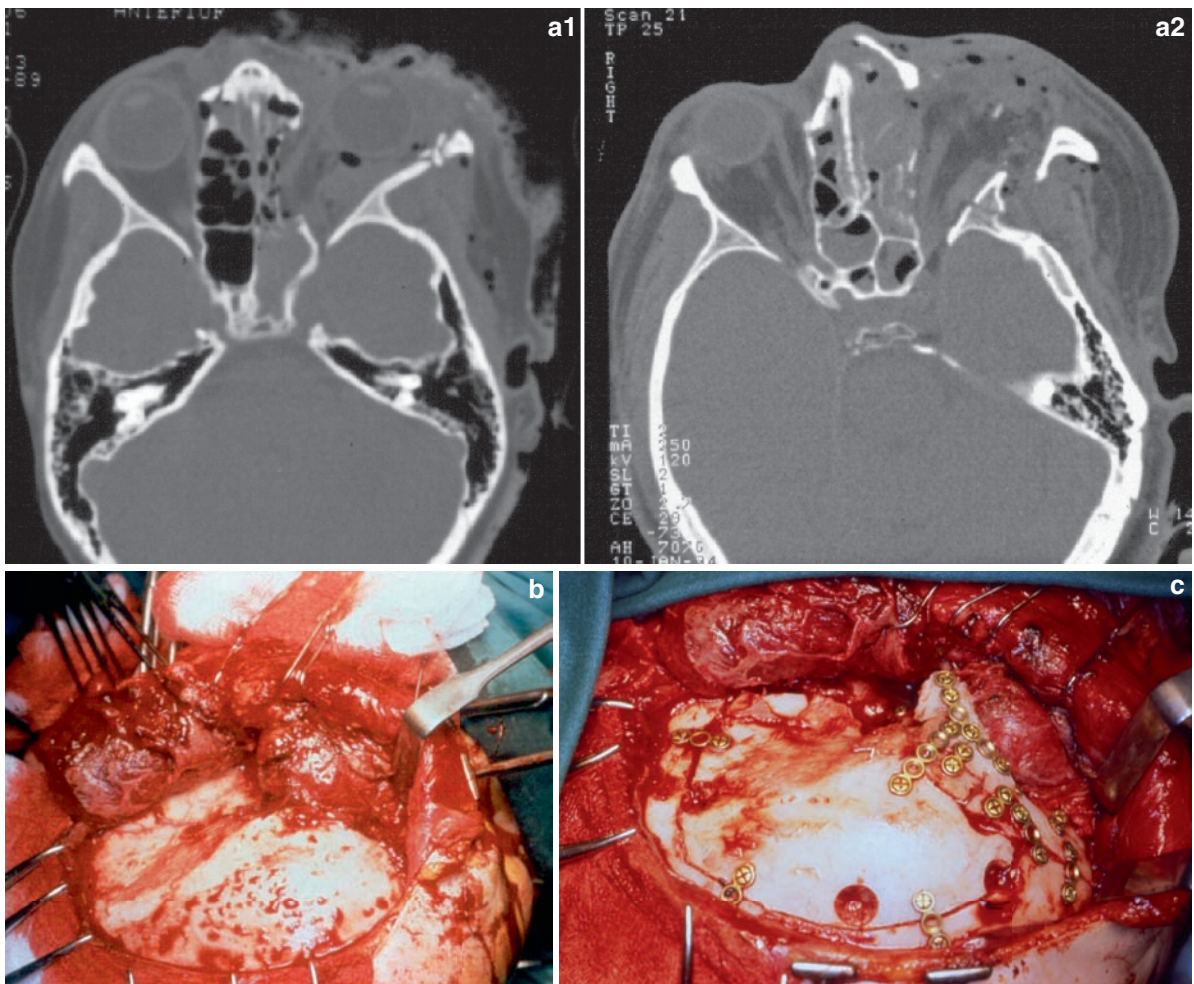


Fig. 12.27 Cranio-orbito-zygomatic-fracture (COF/cranio-orbito-facial fracture). (a) Preoperative CT-scan demonstrating severe displacement of the orbital walls and NOE complex. (b)

Intraoperative view demonstrating comminution of the left lateral orbit. (c) Intraoperative view after craniotomy, dural repair and reconstruction of the zygomatico-orbital complex

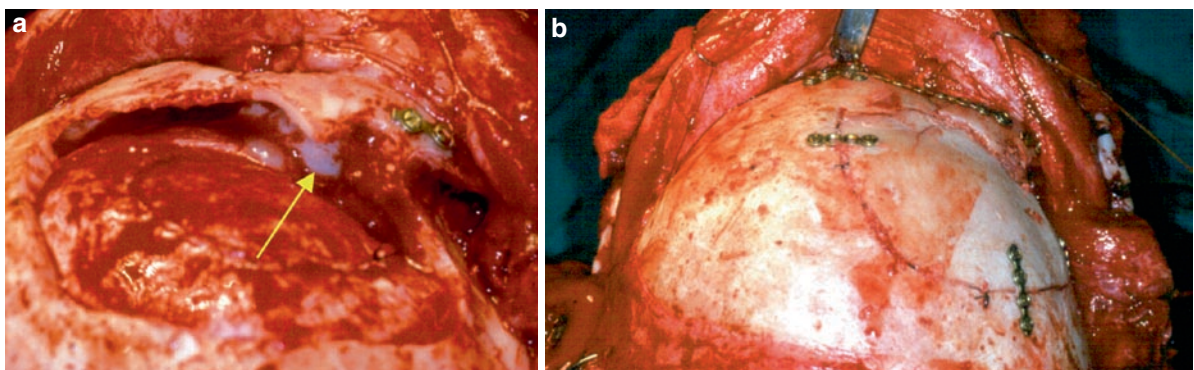


Fig. 12.28 Zygomatico-orbito-cranial fracture with dorsal sinus wall fracture and prolapse of brain tissue (COF). (a) Osteoplastic craniotomy, base exposure and removal of the dorsal sinus wall, intracavitary brain tissue prolapse (arrow). (b)

Reintegration of the bone flap following treatment of the cerebral and dural injuries and reconstruction of the frontofacial and zygomatico-temporal region

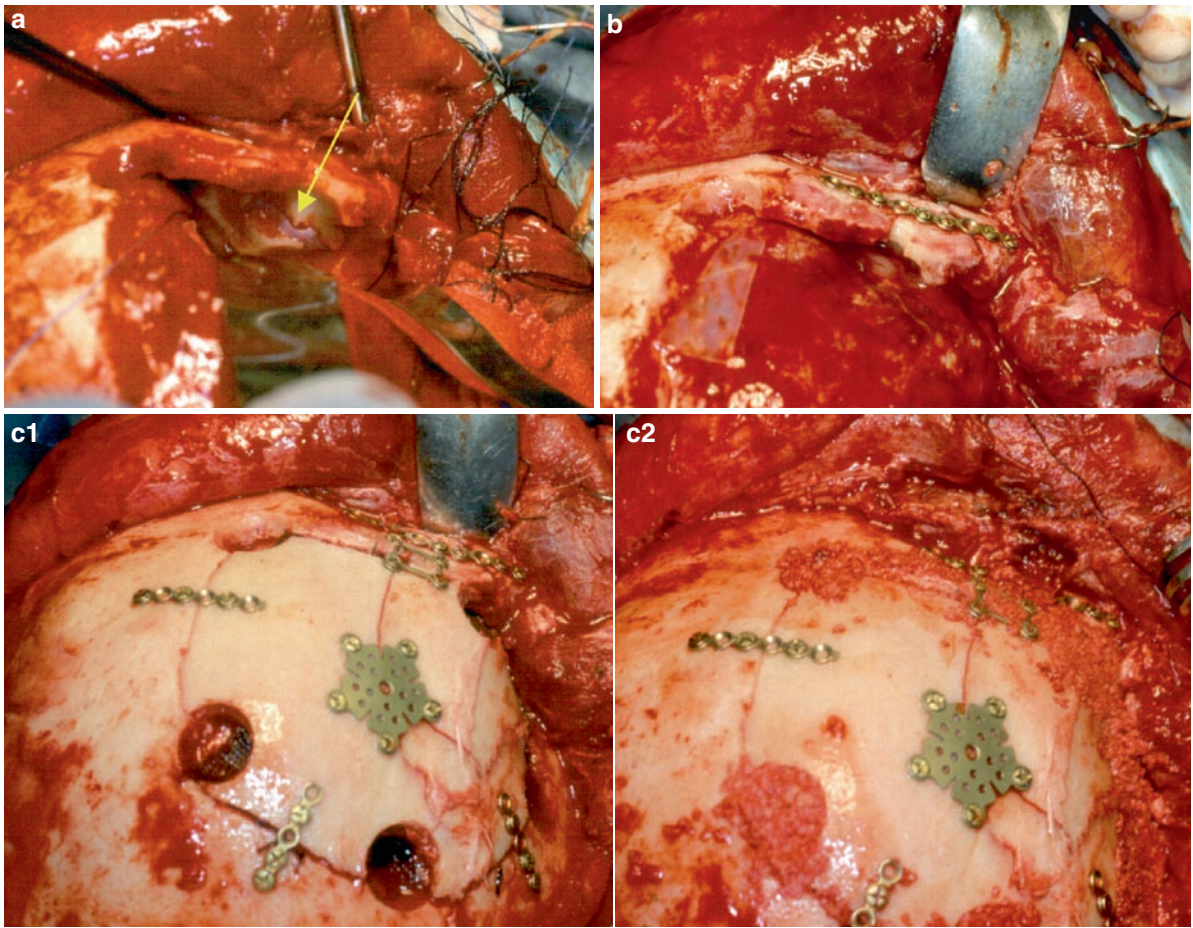


Fig. 12.29 Depressed cranio-orbital fracture with orbital roof fracture (COF). (a) Status following osteoplastic, unilateral craniotomy and exposure of the fractured orbital roof (*arrow*). (b)

Status following orbital roof reconstruction and supraorbital contour restoration. (c) Reintegration of the pre-fixed osteoplastic bone fragments and filling the craniotomy holes with bone dust

12.5.2 Surgical Approach (Baker et al. 1999; Donald 1994; Evans et al. 1996; Baker et al. 2003)

A transfrontal approach is indicated in the following central fronto-cranial injuries:

- Dislocated fractures of anterior and posterior walls of the frontal sinus, limited injuries to the cribriform plate and the medial orbital roof with and without evidence of liquorrhea
- Dislocated fractures of the fronto-orbital complex with contour irregularity, globe displacement and disturbances of eye motility
- Fractures of the fronto-orbital complex with traumatic bone loss of the supraorbital frontal region and the orbital roof

12.5.3 Fractures of the Anterior Frontal Sinus Wall Without Anterior Skull Base Involvement

The surgical approach depends on the extent of anterior wall fracture as well as the degree of dislocation. Either a transfacial or coronal approach is used (Bowles and Anand 1998).

- For anterior wall fractures with minimal to modest degree of dislocation without significant injury to the mucous membranes and intact infundibulum, the fronto-orbital (transfacial) approach may be the one of choice (Theissing 1996; Lappert and Lee 1998).

After exposing the fractures, the impressed anterior wall fragments are raised or temporarily removed (Merville

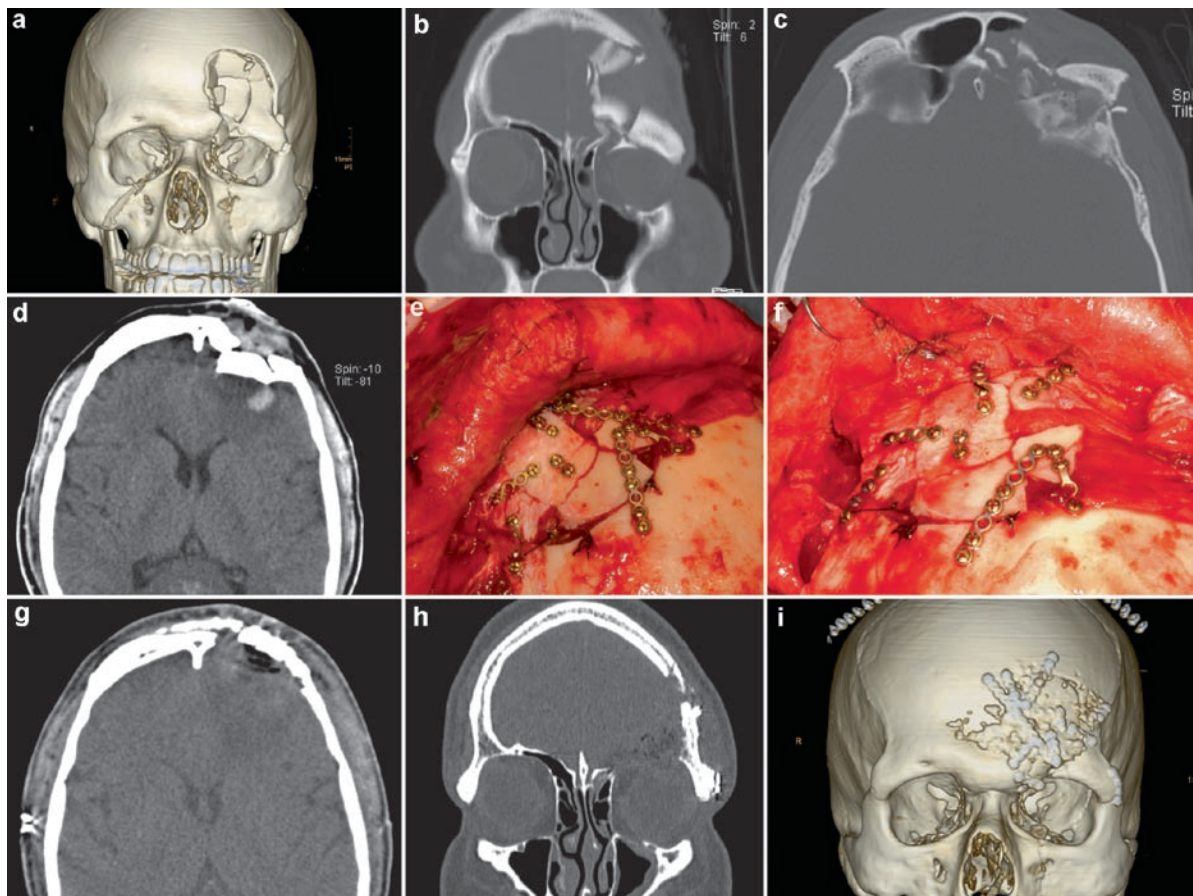


Fig. 12.30 Cranio-orbital fracture (COF). (a–d) Preoperative CT-scan demonstrating depressed comminuted fractures of the fronto-temporal bone and frontal skull base and the orbital roof. (e–f) Intraoperative situation after reconstruction of the frontal skull base, dural repair, cranialization of the frontal sinus,

insertion of a pericranial flap and fixation of the frontal bone fragments. g–i Postoperative situation after frontal craniotomy and cranialization of the frontal sinus and reduction and fixation of the cranial fragments with multiple 2.0 mm miniplates

et al. 1983; Mathog 1984; Hoffman and Krause 1991). Small bony fragments, foreign bodies and lacerated portions of the mucous membrane are carefully removed from the frontal sinus. The posterior wall of the frontal sinus is carefully examined (endoscopically) for additional fractures. The patency of the nasofrontal duct is examined using methylene blue. All bony fragments are replaced in the exact anatomical position and fixed with mini- or microplates (Prein 1998). Even small fragments can be used for reconstruction (Figs. 12.31–12.34).

Residual frontal defects, larger than 1 cm² should be reconstructed. This can be carried out using autogenous bone grafts (calvarial grafts – tabula externa) or alternatively, in particular in anatomically difficult regions with titanium micromesh (Dempf et al. 1998;

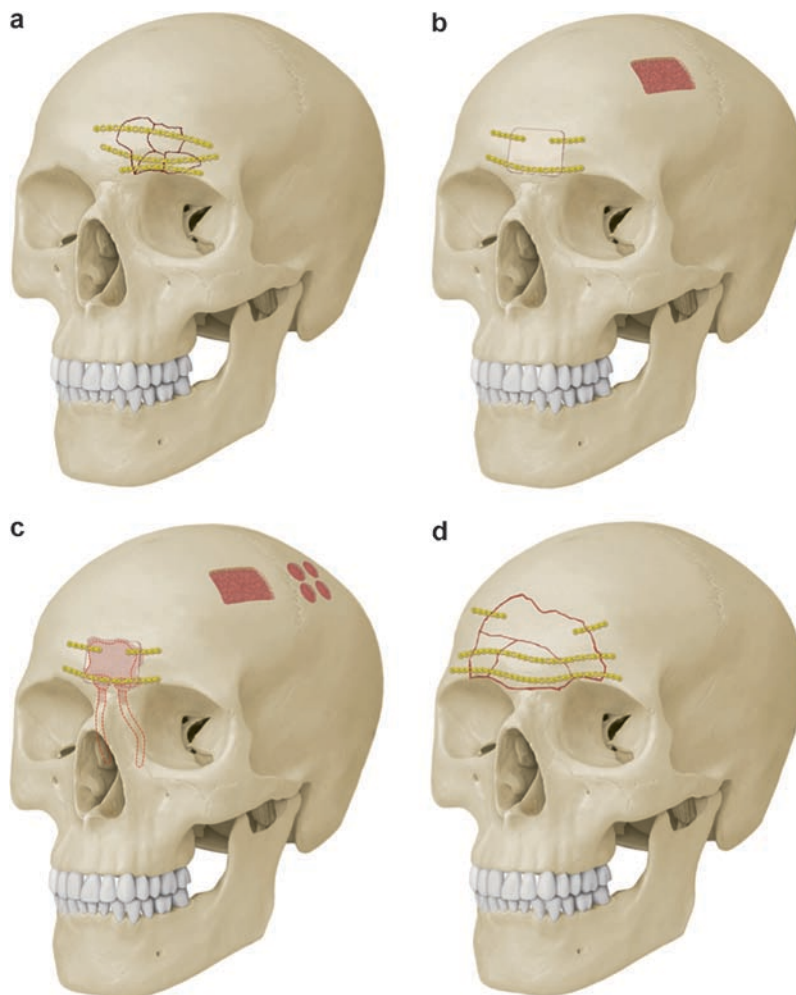
Vesper et al. 1998; Deinsberger et al. 1998; Kuttenger and Hardt 2001) (Fig. 12.35).

Disadvantages of the fronto-orbital approach are the limited surgical exposure and visible scars. Limited scars in the naso-orbital region can be concealed by the eyebrows and should be placed in natural skin lines.

- For extensive, depressed and comminuted fractures of the anterior wall of the frontal sinus the coronal approach is recommended.

In general, drains have to be placed in all fractures affecting the paranasal sinuses. In fractures involving the frontal sinus, sufficient ventilation and drainage can be achieved by resection of the interfrontal septum

Fig. 12.31 (a) Reconstruction of the anterior wall of the frontal sinus with miniplates or microplates. (b) Reconstruction of the anterior sinus wall with a split skull graft taken from the outer table in case of excessive comminution. Fixation with 1.3/1.5-mm miniplates or microplates. (c) Obliteration of the frontal sinus in comminuted anterior wall fractures: complete removal of the mucosa, obliteration of the nasofrontal duct with a bone graft and filling the sinus with cancellous bone from the parietal region. The anterior table is reassembled and stabilized with 1.3/1.5-mm mini- or microplates. (d) Cranialization of the frontal sinus (fracture of the anterior wall, comminution of the posterior wall). Removal of the posterior wall and reconstruction of the anterior wall. The floor of the frontal sinus and the floor of the anterior cranial fossa are covered with a pericranial flap and occasionally bone grafted with cancellous or calvarial bone grafts (mod. a. Prein 1998)



and a high nasal septum. Intranasal drains can also be placed into the sinus through the frontal infundibulum (Richter 1992).

12.5.4 Fractures of the Posterior Frontal Sinus Wall with Anterior Skull Base Involvement

Small dural defects caused by fractures of the dorsal wall of the frontal sinus and dural defects at the junction to the ethmoid and cribriform plate can in some cases be directly occluded with sutures. Additional dural repair should be performed with autogenous fascial grafts or alloplastic dural substitutes (e.g., Neuropatch) (Ernst et al. 2004).

More extensive dural defects are closed with dural substitutes (e.g., Neuropatch, pericranial grafts, fascia lata) after the dura has been released from the bony margins.

All dural grafts and membranes are either fixed with sutures or fibrin glue (Stoll 1993; Ernst et al. 2004). After dural closure in selected cases the posterior sinus wall can be reconstructed using the bone fragments available (Hybels 1977; Elies 1982; Stoll 1993). If extensive dural injuries exist beyond the region of the frontal sinus (ethmoid, medial orbital roof) the transfrontal approach has to be used (Donald 1998a).

- In sinus wall fractures with additional frontal skull base injuries, the transfrontal approach with a transcranial-extradural or subcranial-subdural exposure is favored due to aesthetic and surgical reasons

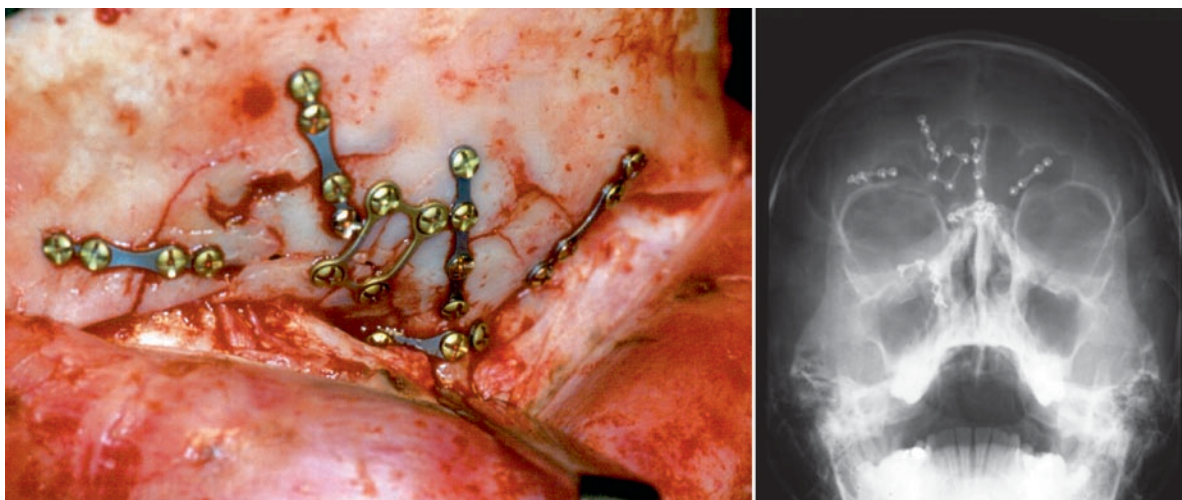


Fig. 12.32 Reconstruction following dislocated anterior wall fracture of the frontal sinus, including naso-maxillary fracture on the right [comminuted craniofrontal fracture (CCF)]. Fixation of multiple bone fragments with microplates

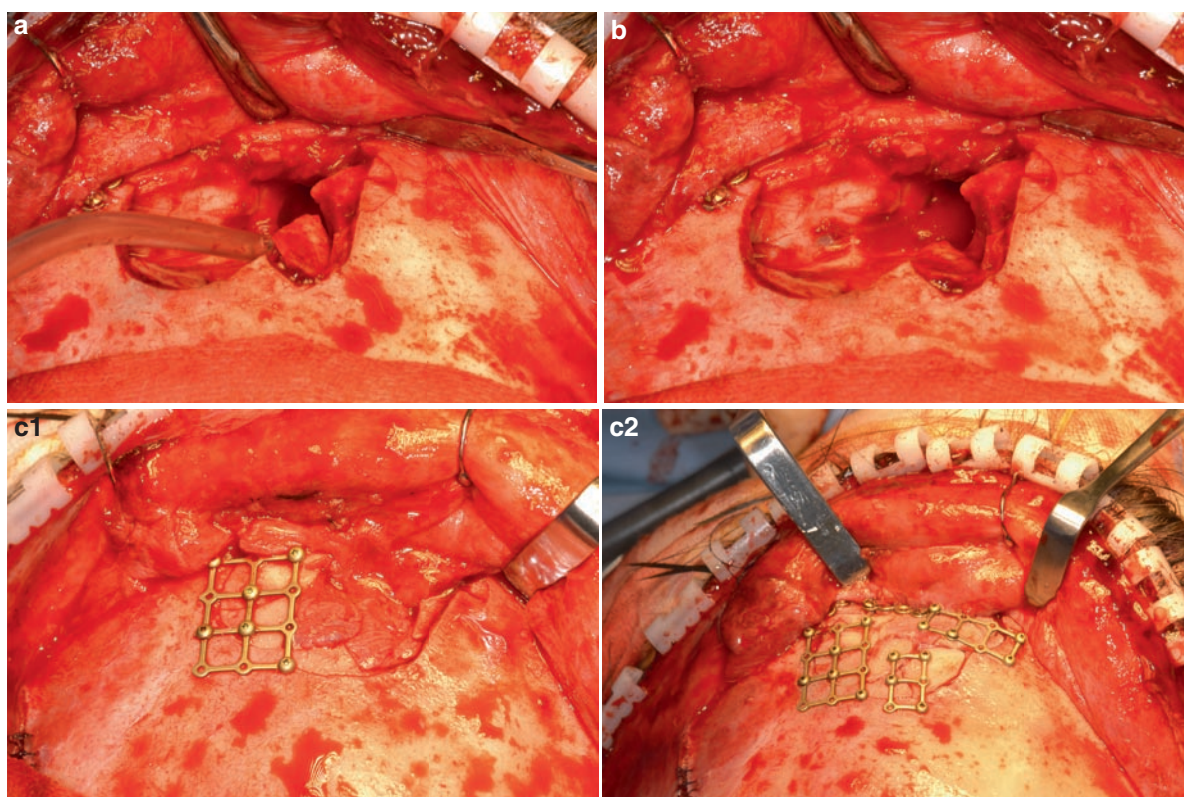


Fig. 12.33 Anterior frontal sinus wall fracture with depressed fragments (CCF). (a) Exposure via coronal approach — fragment removal. (b) Examination of dorsal sinus wall and nasofrontal

duct. (c) Reconstruction starting from the fracture margin, stabilization with three-dimensional miniplates

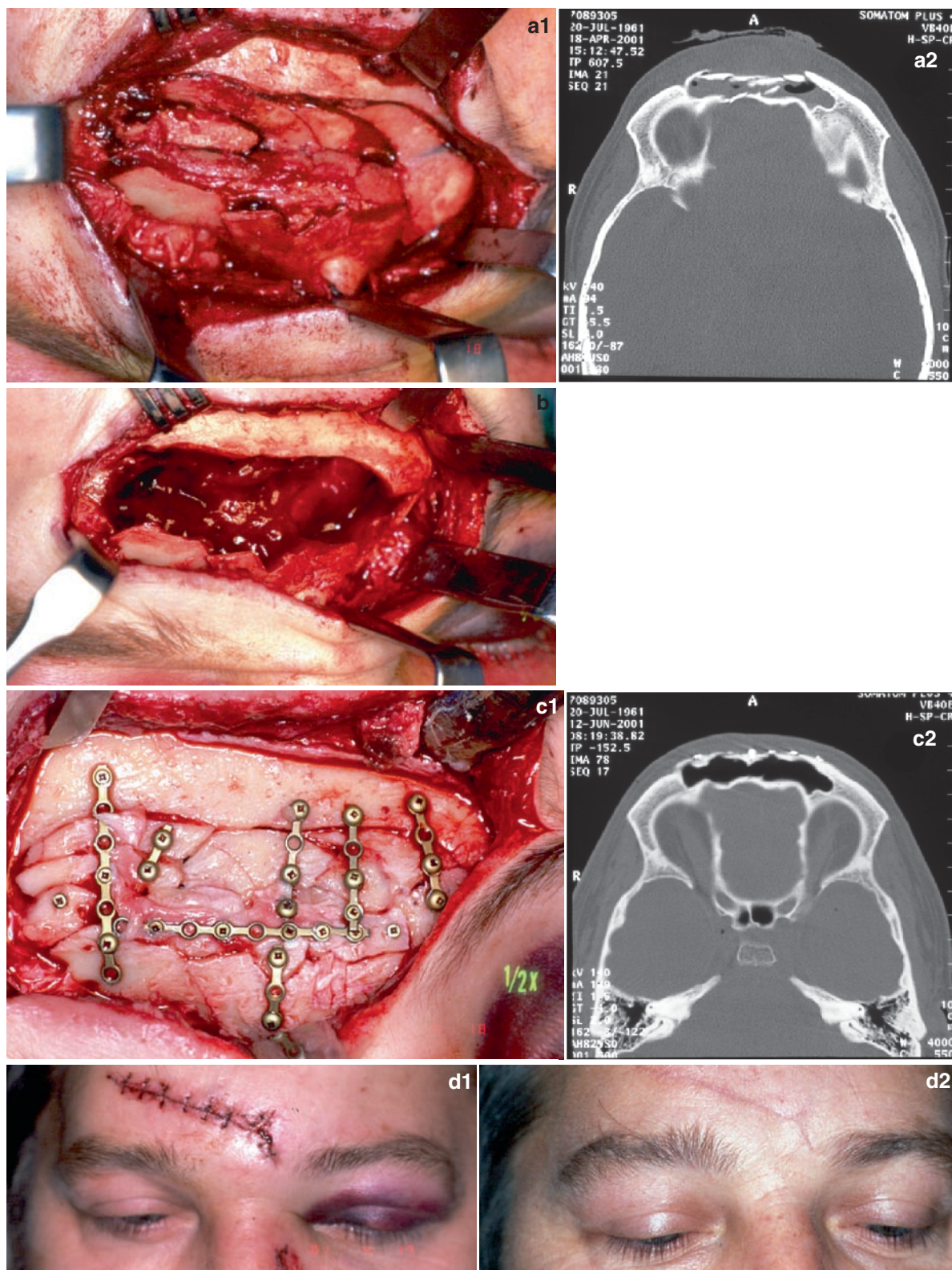


Fig. 12.34 Comminuted anterior sinus wall fracture (CFF). (a) Comminuted anterior sinus wall (following transfacial fracture exposure/X-ray). (b) Removal of the bony fragments. (c)

Reduction and osteosynthesis of the anterior sinus wall (clinically/X-ray) with 1.5-mm miniplates. (d) Postoperative result (1 week/3 months postoperatively)

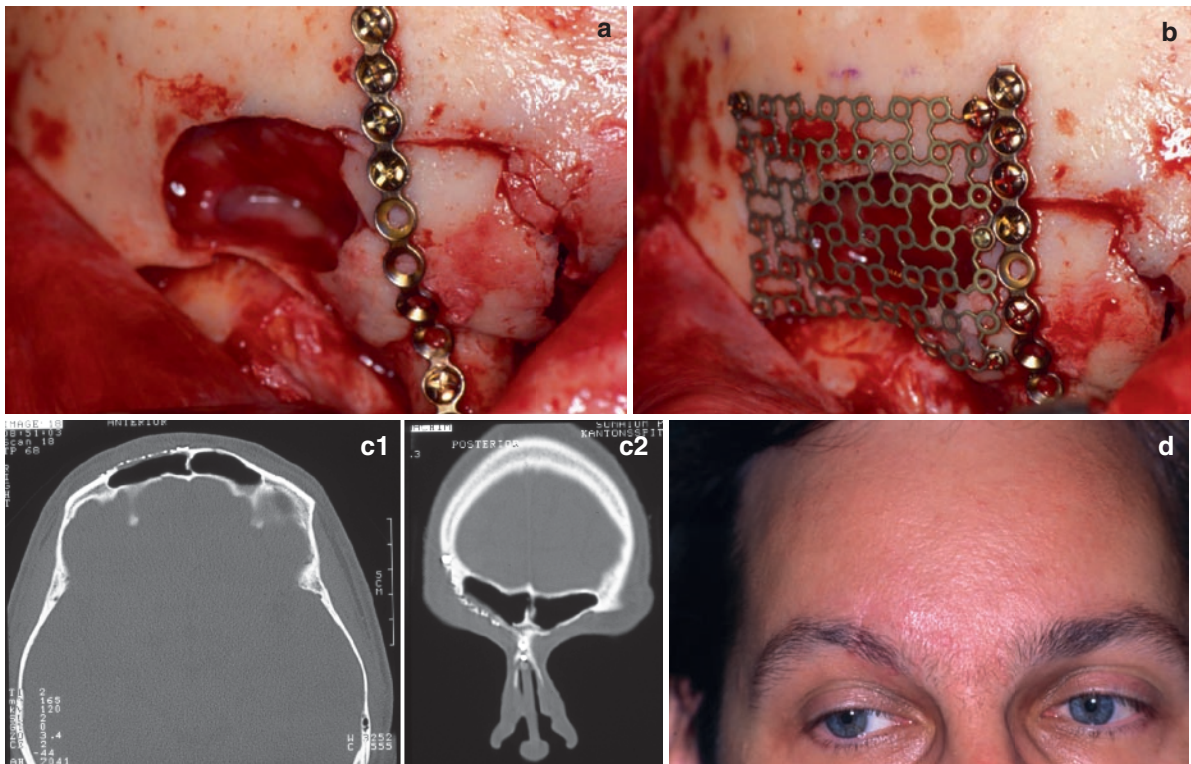


Fig. 12.35 Reconstruction of the anterior sinus wall with titanium mesh (CFFs). (a) The larger bone fragments are used to reconstruct part of the anterior wall of the frontal sinus. Multiple small fragments cannot be fixed resulting in a bone defect. (b) Closure of the defect with a dynamic titanium mesh (0.3 mm).

(c) CT scans 6 months postoperatively demonstrating a symmetric frontal contour and complete re-pneumatization of the frontal sinus. (d) Clinical situation 6 months postoperatively, demonstrating smooth and symmetric contour of the supraorbital region

12.5.5 Fractures of the Anterior and Posterior Sinus Walls with Anterior Skull Base Involvement

Basically, the principle aim is always to reconstruct the frontal sinus (Ehrenfeld et al. 1996; Ernst and Lennarz 1996).

In extensive frontal sinus fractures with frontal skull base injuries, the transfrontal/transcranial extradural approach should be favored. Following temporary removal of either the anterior wall of the frontal sinus or a cranio-frontal bone flap, the dura and base are exposed. Alternatively, the affected skull base section can be approached and treated via the subcranial/subdural approach (Raveh and Vuillemin 1988; Raveh et al. 1992).

In the case of extensive comminution, particularly of the dorsal wall, *cranialization* of the frontal sinus is performed by total removal of the dorsal wall, meticulous removal of the sinus mucosa, following tight closure of

the infundibulum. This results in a functional enlargement of the intracranial space (Kessel et al. 1971; Donald and Bernstein 1978; Luce 1987; Elies 1982; Wallis and Donald 1988; Donald 1994, 1998a, b) (Figs. 12.36–12.39).

- Occlusion of the nasofrontal duct is carried out with a bone or muscle graft and an additional pericranial flap. The ventral sinus wall should be always reconstructed.

12.5.6 Fractures of the Frontal Sinus with Comminution of the Infundibulum

Ventilation and drainage of the frontal sinus may be difficult to restore in fractures with intact posterior sinus wall and simultaneous comminution of the anterior wall and infundibulum. In such cases, *obliteration* of the sinus with autogenous tissue (bone graft, fat, and muscle) is indicated (May et al. 1970; Stanley and Becker 1987;

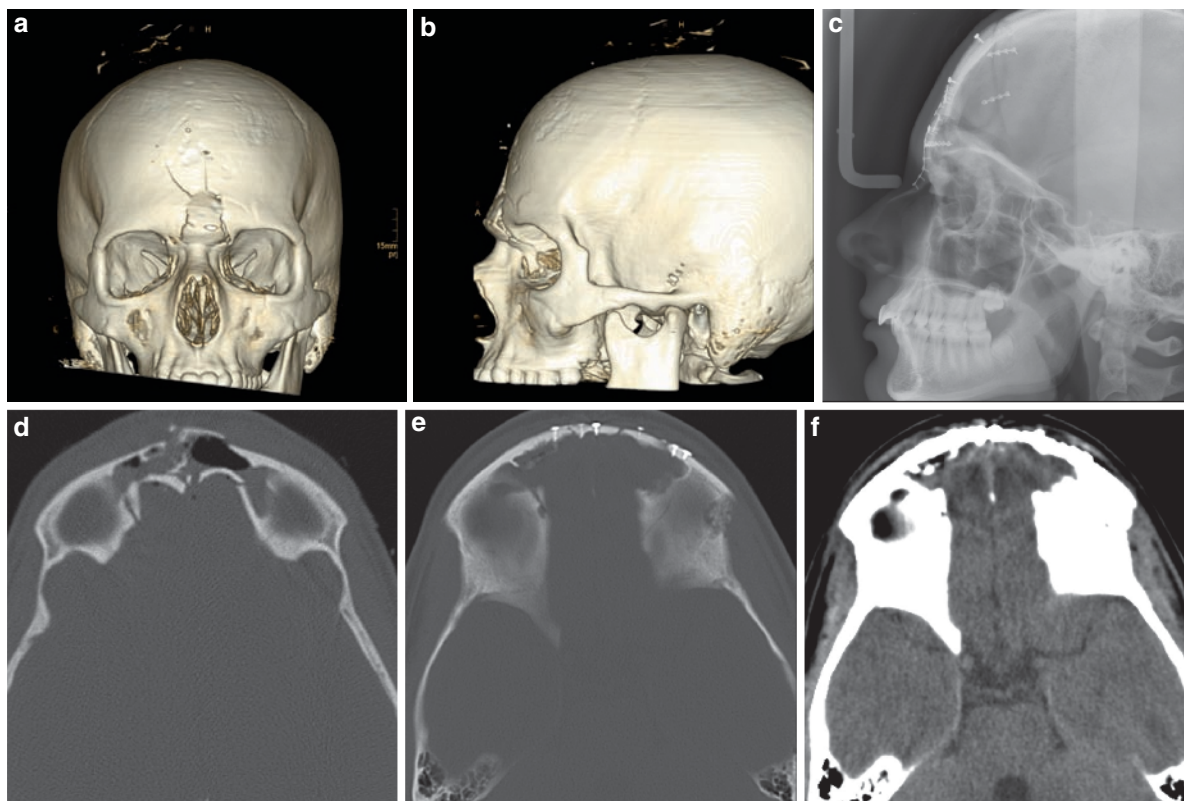


Fig. 12.36 Cranialization of the frontal sinus after localized comminuted craniofrontal fracture (CFF). Initial CT (**a**, **b**, **d**) and postoperative result (**c**, **e**, **f**) with perfect reconstruction of

the facial contours (**c**), resection of the posterior sinus wall and dural reconstruction (**f**). Residual post-contusional defects in the frontal lobe

Donald and Ettin 1986; Wilson et al. 1988; Ehrenfeld et al. 1996; Sailer et al. 1998; Ernst et al. 2004).

Principle of frontal sinus obliteration

- Total removal of the sinus mucosa
- Careful removal of the inner cortical layer with the diamond burr
- Posterior wall inspection regarding fractures and dural integrity. In fractures without dural injury, the posterior wall should additionally be covered with a membrane or an autogenous graft (pericranium, fascia). In the case of bony defects, the bone fragments can be reintegrated
- Nasofrontal duct occlusion with a cortico-cancellous bone graft
- Filling the sinus with condensed cancellous bone or with crushed cortico-cancellous bone chips
- Anterior sinus wall replacement, screw fixation or coverage with a bone graft (e.g., tabula externa graft)

Whereas several authors recommend muscle or fat grafts for obliteration, today the use of condensed, autogenous cancellous bone from the iliac crest is favored (Hausamen and Schierle 2000). After thorough homogenization using a bone mill, the bone graft is introduced into the frontal sinus and condensed there.

For stability reasons, the anterior sinus wall has to be reconstructed either with a calvarial bone graft (Prein 1998) or by applying titanium mesh (Kuttenberger and Hardt 2001). If the frontal sinus is obliterated with cancellous bone, a 0.3-mm mesh is recommended for stability reasons and to prevent contour irregularities due to bone resorption.

12.6 Own Statistics

In 61% of combined comminuted fractures of the anterior and posterior walls, the posterior sinus wall was removed and a cranialization with simultaneous anterior

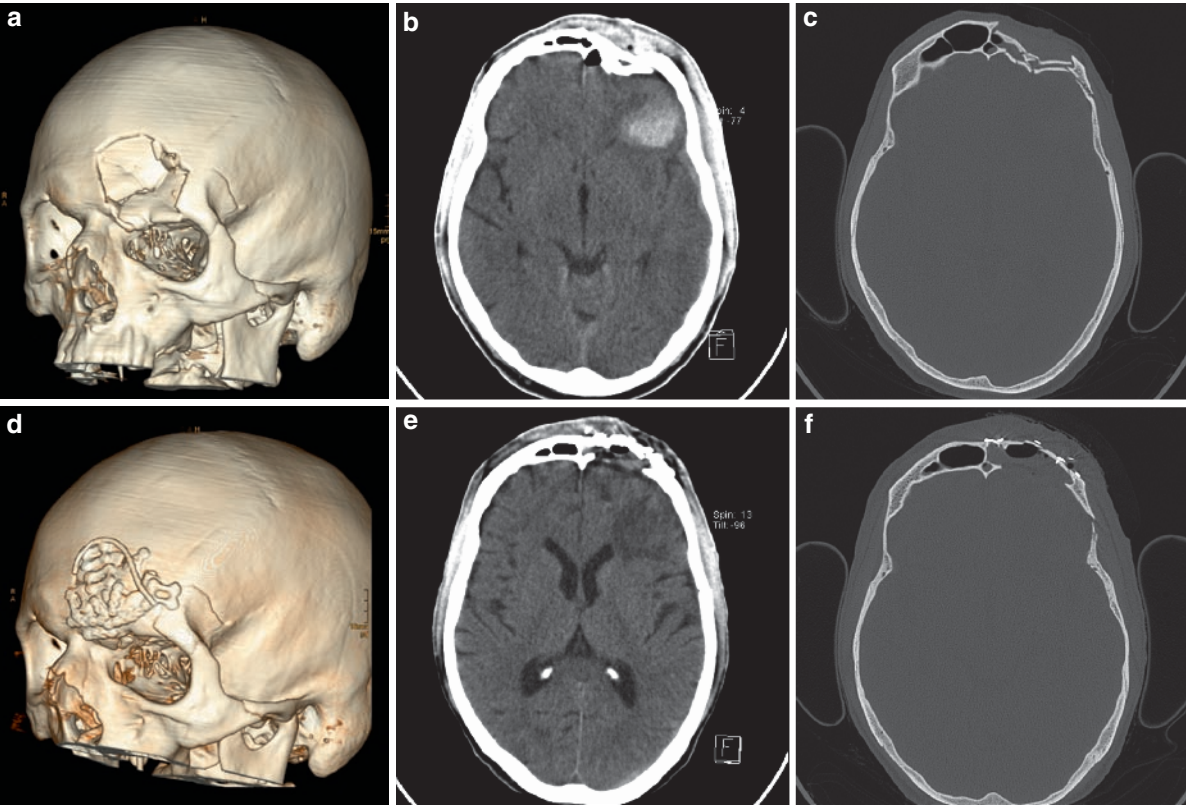


Fig. 12.37 Comminuted craniofrontal fracture (CFF) with brain injury. (a–c) Impression fracture of the frontal bone with contusional hemorrhage in the left frontal lobe. Fracture of anterior and posterior walls of the frontal sinus and downward displacement

of the orbital roof. (d–f) Postoperative result. Reduction and fixation of the frontal bone fragments with excellent restitution of the contours (d). Resection of the posterior wall of the left frontal sinus (cranialization) and reconstruction of the dura (e, f)

sinus wall reconstruction and obliteration of the nasofrontal duct were carried out. After total removal of the sinus mucosa, the nasofrontal duct was sealed off with a muscle patch from the temporal muscle and a frontal pericranial flap.

In 29% of isolated anterior wall fractures, reconstruction was performed with local bone and mini-plates (24%); in 5%, titanium meshes were used.

Obliteration of the frontal sinus with cancellous bone was performed in 7%. In all cases of obliteration, the posterior sinus wall was maintained and the anterior wall reconstructed with a calvarial graft (outer table).

Simultaneous reconstruction of the anterior and posterior wall (without dural injury) was carried out in 3% (Neidhardt 2002).

Techniques for treating frontal sinus fractures (Neidhardt 2002)	
Cranialization and anterior wall reconstruction	61%
Anterior wall reconstruction	29% (24%/5%)
Obliteration and anterior wall reconstruction	7%
Anterior and posterior wall reconstruction	3%

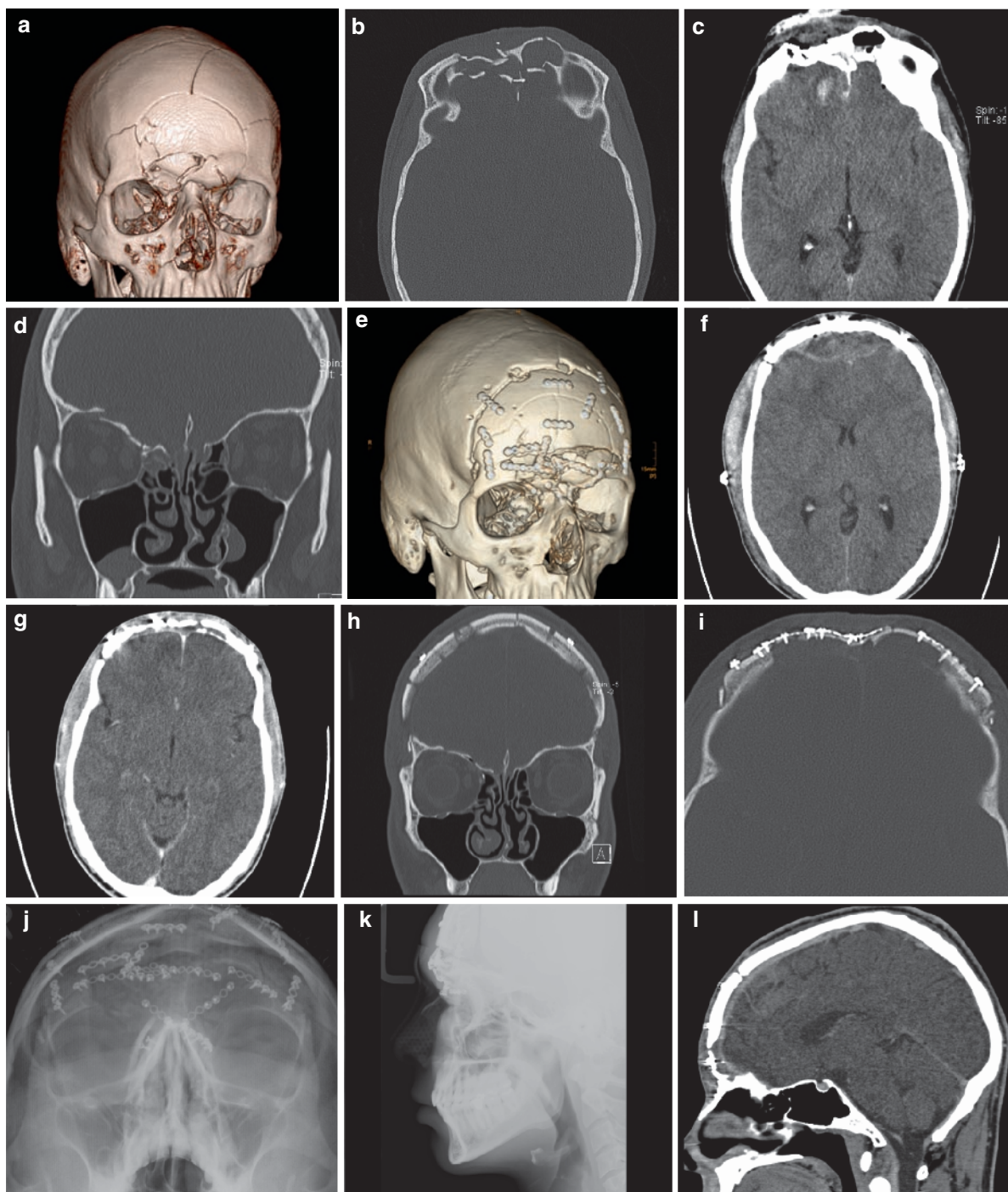


Fig. 12.38 Multifragmentary fronto-cranial fracture with CFF. (a–d) Initial CT scan demonstrating comminuted fractures of the nasofrontal complex (a, b), frontal sinus (b, d) and right orbital roof (d). Frontal brain hemorrhage on the right side (c). (e–l) After

reconstruction of the frontal skull base, dural repair and cranialization of the frontal sinus (f, g, l), insertion of a pericranial flap and fixation of the frontal bone fragments with several mini-plates (e, i, j). Excellent restitution of the frontofacial contour (k, l)

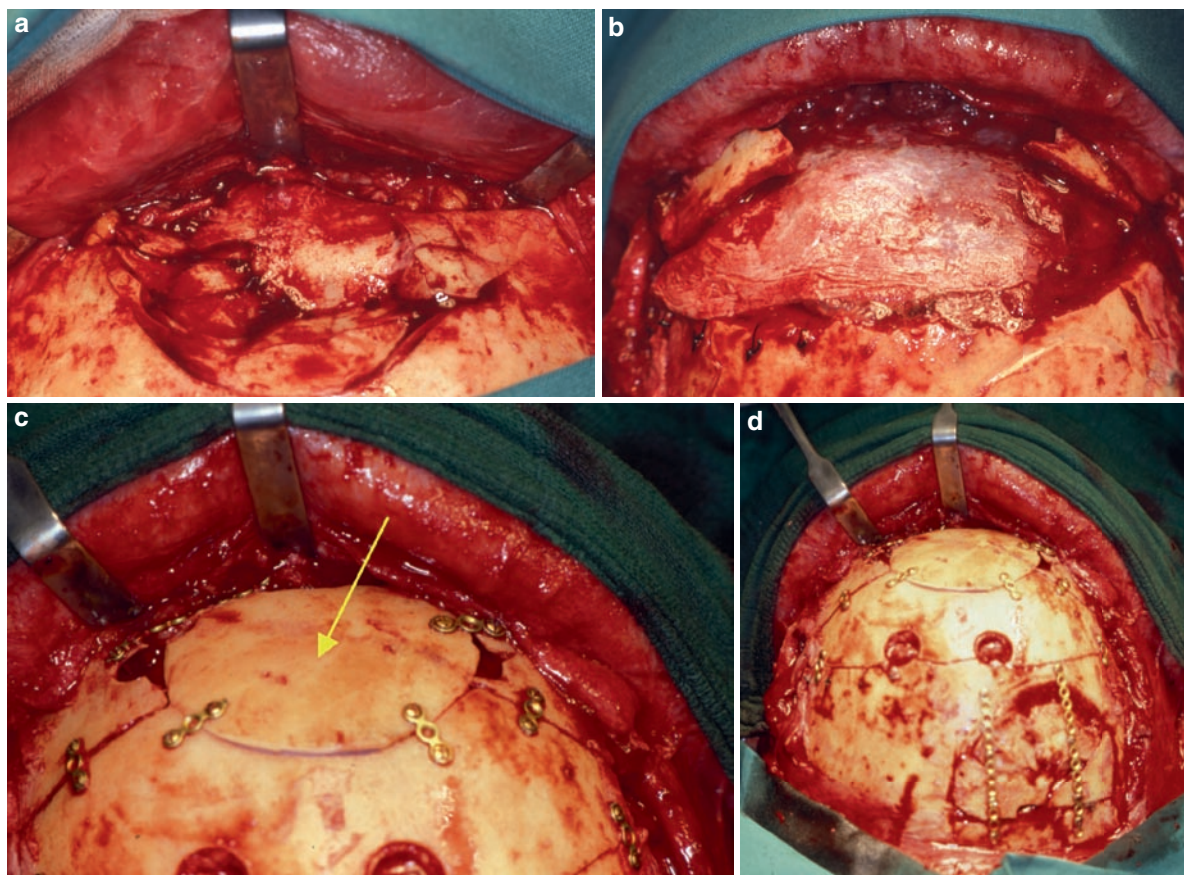


Fig. 12.39 Reconstruction of frontal region with split calvarial graft. (a) Comminuted fracture of the frontal bone (CFFs). (b) Status after removal of all bony fragments, including anterior and posterior frontal sinus wall and dural repair with fascia. (c)

The anterior sinus wall and part of the frontal bone is reconstructed with a monocortical bone graft (tabula externa) (arrow) harvested from the parietal region. (d) The donor side is filled with the small anterior bone fragment

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The early and late complications following treatment of severe craniofacial injuries cannot always be differentiated from those of the injury itself. Complications may develop with or without and despite or because of a surgical intervention (Sprick 1988; Schmidek and Sweet 1988).

Complications which develop within a short period of time after the injury (<1 month) are classified as early complications. Late complications develop after an interval of 2-3 months (Hardt and Steinhäuser 1979).

Postoperative complications are dependent on various modalities:

- Severity of the injury
- Time of treatment
- Quality of treatment
- Absence of primary treatment

Postoperative complications

Postoperative complications cause specific problems (Kretschmer 1978; Schmidek and Sweet 1988), such as:

- Subgaleatic, epidural, subdural, intracerebral abscesses
- Epidural-subdural hemorrhage/hematoma
- Osteomyelitis of replanted bone fragments or autogenous bone grafts
- Hygroma
- Sinus complications
- Recurrent liquorrhea
- Elevated intracranial pressure

Own statistics

Our own postoperative early and late complications following treatment of craniofacial fractures are distributed as follows (Neidhardt 2002):

Postoperative complications and late sequelae following craniofacial reconstruction

Recurrent liquorrhea	2.8%
Infections	11%
Osteomyelitis	4%
Epi-/subdural hematoma	3%
Subdural Hygroma	2%
Mucocele of frontal sinus	2%
Functional neurological deficits	16%
Olfactory nerve deficit	8%
Contour irregularities	8%
Letality	6%

13.1 Infections and Abscesses

The postoperative complications in our group of patients proved to be relatively low. The most common complication was infection (15%). These infections can be divided into:

- Localized (early) infections: subgaleatic, epidural, abscesses/empyemas
- Late infections: osteomyelitis

Localized infections were observed in 11%. *Staphylococcus aureus* could be detected in most of these of cases:

- 3% subgaleatic infections
- 4% epidural abscesses
- 4% infected seromas

The infected seromas were found in the temporal area away from the actual fracture site and were caused by harvesting large temporal muscles patches. The epidural abscesses resulted from epidural dead space caused by failing expansion of the brain.

Subgaleatic and epidural abscesses require urgent revision of the operation site with facultative removal

Fig. 13.1 Subgaleatic infection. (a) Infection following insufficient transfacial primary treatment. (b) State following craniotomy. Removal of infected fragments and osteosynthesis material, fronto-facial-osteotomy, and skull base revision. (c) Reconstruction of the frontal region. Closure of the remaining defect with bone dust and titanium mesh. (d) Postoperative situation

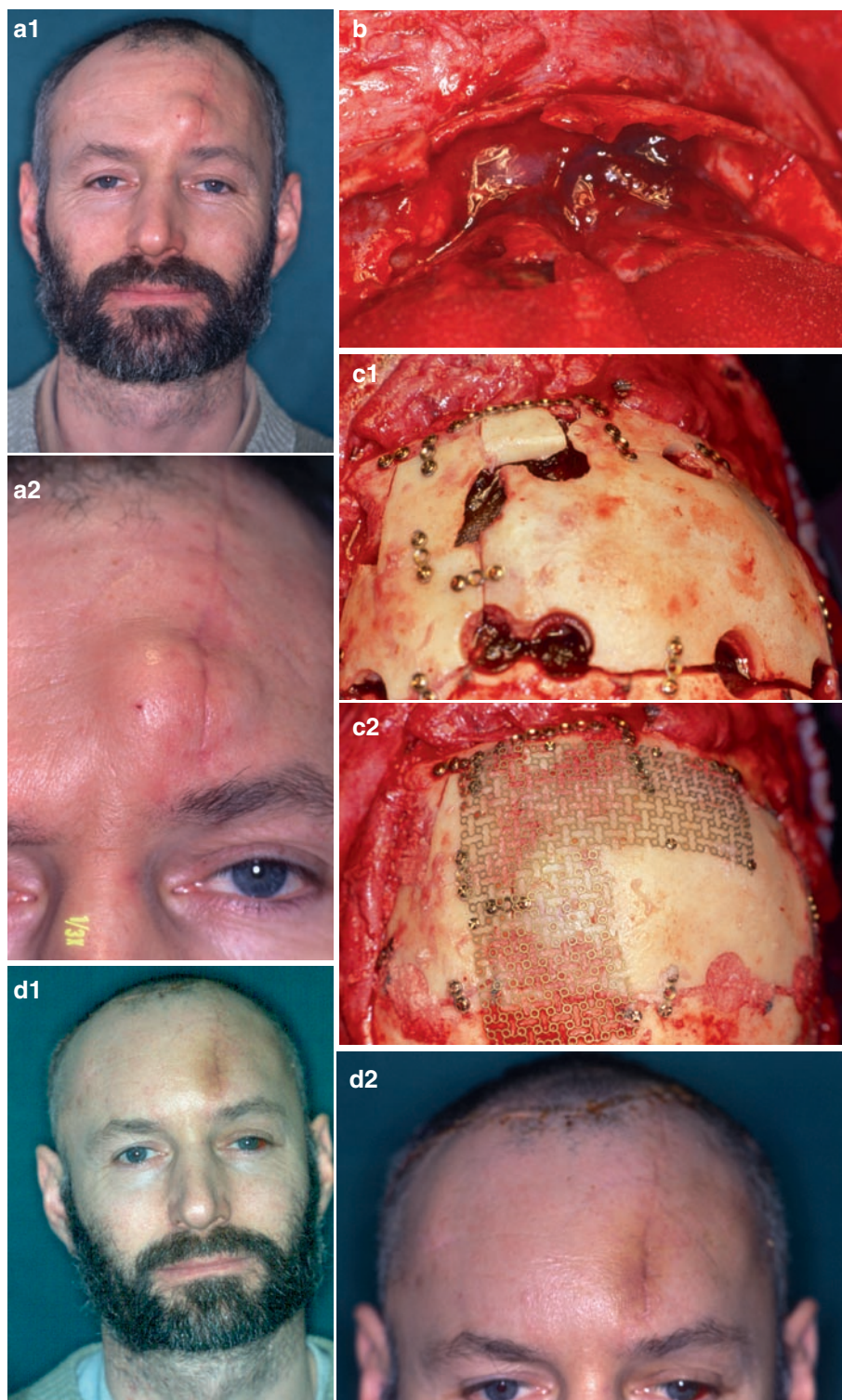
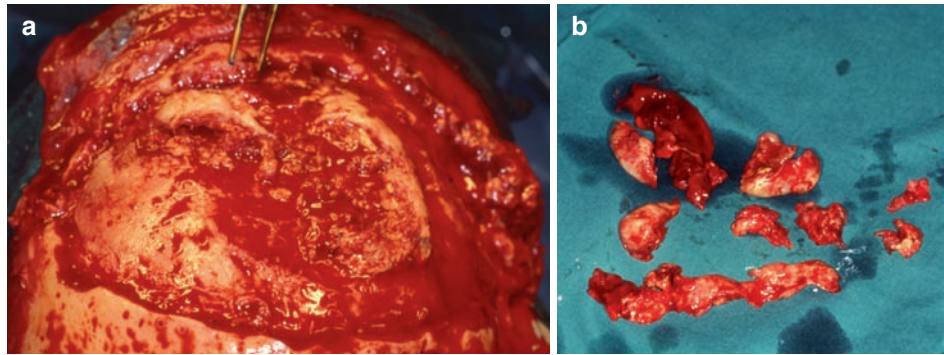


Fig. 13.2 (a) Chronic epidural abscess after temporary removal of the bone flap and debridement of the infected fragments. (b) Specimen of the abscess membrane



of the osteosynthesis material and obligate removal of the infected bone fragments, a selective antibiotic therapy and drainage (Figs. 13.1 and 13.2).

13.2 Osteomyelitis

Despite adequate reconstruction and sufficient perioperative antibiotic prophylaxis, infection or necrosis of local bone fragments or grafts may occur. Osteomyelitis can develop with varying latency in areas of insufficiently revitalized bone.

Besides general signs of infection and local inflammatory changes of the galea, there is radiological evidence of a permeative osteolysis of the bone. If recurrent swelling, pain, and fistulae occur, the operation site has to be revised due to the vital danger of osteomyelitis.

In 4% of our own patients, osteomyelitis was the cause for revision of the frontofacial operation site:

- Osteomyelitis of local bone (2%)
- Osteomyelitis of bone grafts (2%)

Therapy consists of removing the osteosynthesis material from the infected and neighboring areas and removing the affected bone (sequestrotomy) until vital bony margins are found. The dural reconstruction has to be examined.

Bony reconstruction can be postponed and a titanium mesh inserted for immediate contour restoration. Immediate reconstruction with cancellous bone and titanium mesh, however, also proved to be successful (Esser and May 1990; Kuttnerberger et al. 1996) (Figs. 13.3 and 13.4).

13.3 Recurrent Liquorrhea

The most common complication following skull base treatment in combination with midface fractures is

persisting or recurrent liquorrhea. The following causes may be responsible (Boenninghaus 1974; Kretschmer 1978; Myers and Sataloff 1984; Probst 1986; Brachvogel et al. 1991):

- Insufficient repair of dural defects
- Displacement of dural reconstruction (grafts, membranes) through manipulation (e.g., midface reduction following skull base treatment)
- High intracranial pressure
- False diagnosis of skull base fractures with dural injuries
- Iatrogenic dural injuries

To detect the reasons for recurrent liquorrhea, specific radiograph examinations are necessary (CT/Jotrolan-CT/MRI/liquor scintigraphy). To locate persisting liquor fistulae, intraoperative liquor marking with sodium fluorescein has proved its value (see Chap. 8).

The frequency of recurrent liquorrhea not only depends on the surgical technique but also on the type of injury. According to the modern literature it varies between 2% and 6% (Probst 1986; Lange et al. 1995).

Recurrent liquorrhea following dural and skull base repair (Donald 1998)

Ketchham et al. 1963**	25%
Probst 1971*	9%
Loew 1984	6%
Probst 1986	6%
Bergemann et al. 1993**	5%
McCutcheon et al. 1993**	6%
Kraus et al. 1993**	2%
Deschler et al. 1994**	11%
Lange et al. 1995	3%
Schramm 1997**	3%
Neidhardt 2002	3%
Lädrach 2007	2.3%

* cited by Probst 1971 - see the reference further down

** cited by Donald P. J., 1998 - see the reference further down

No star – These authors are cited on their own in the reference list

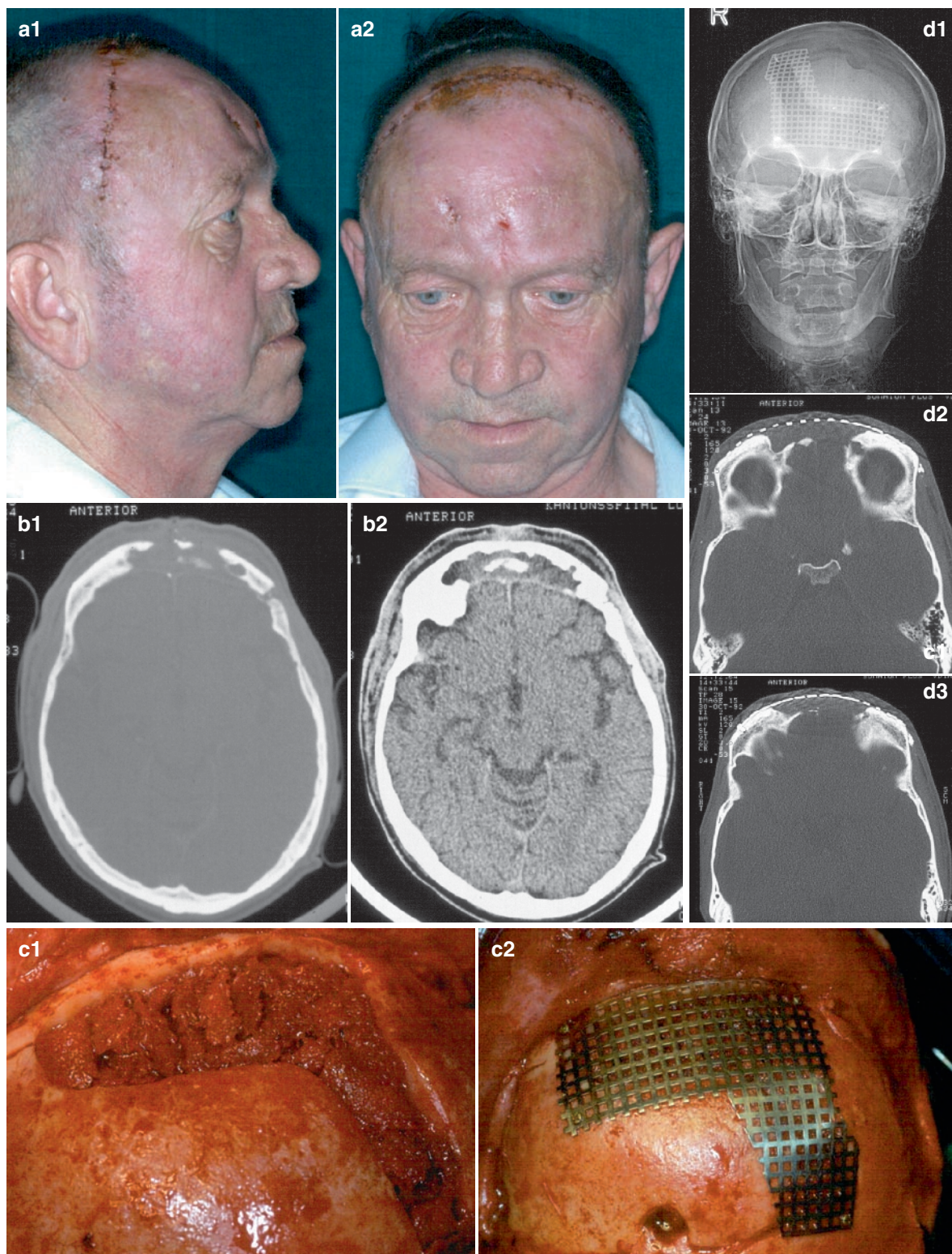


Fig. 13.3 Frontal osteomyelitis. Reconstruction of the cranio-frontal region with cancellous bone and titanium mesh. (a) Frontal cutaneous fistula formation following frontofacial reconstruction. (Gun shot injury of the midface and frontal skull

base.) (b) CT: osteolysis of the frontal bone with sequestration. (c) Reconstruction with cancellous bone graft and titanium mesh after sequestrotomy. (d) Postoperative X-ray and CT control

Raveh and Vuillemin (1988) found recurrent liquorrhea in only 1.9% of 374 patients treated for cranio-facial fractures. The number of preoperative cerebrospinal fluid (CSF) rhinorrheas of patients operated on within the first 24 h, however, was unknown (Probst and Tomaschett 1990).

Sealing of dural lacerations has become more successful with the introduction of fibrin glue and the use of fascia-lata grafts (Raveh et al. 1984, 1988; Probst and Tomaschett 1990; Ladrach 2007) found postoperative CSF leakage in 2.3% of craniofacial traumas with skull base and dural injuries.

In our patient group, recurrent liquorrhea occurred in 2.8% of all operated patients. In the remaining patients, neither immediate postoperative nor late liquorrhea was found.

This proves that our surgical technique, which includes wide exposure, multi-layered dural and skull base repair and primary bony reconstruction provides reliable and stable results.

Our treatment concept for recurrent liquorrhea initially consists of waiting for 2-3 weeks with simultaneous antibiotics, 30° head inclination and lumbar drainage. If no spontaneous occlusion of the fistula occurs, localization of the leakage is performed with the use of CT/Jotrolan - CT/MRI/Liquor Scintigraphy/Na-Fluorescein liquor marking.

Treatment concept for recurrent liquorrhea:

- Wait 2-3 weeks/antibiotics/30° head elevation/lumbar drainage
- Clarify fistula localization
- (Coronal CT/Jotrolan-CT/MRI/liquor scintigraphy/Na fluorescein)
- Frontal skull base revision

Depending on the localization of recurrent liquor fistulas, either the transfrontal or the endonasal approach is performed. Occasionally a combined procedure is necessary.

In extreme cases with delayed liquorrhea and extensive bone loss, the skull base is either reconstructed with bone grafts or titanium mesh. The reconstructed skull base has to be covered with local flaps (pericranial flaps) or, alternatively, occluded by integrating microsurgically anatomized free flaps (fascio-cutaneous flaps) (Seeger 1983; Bootz and Gawlowski 1995; Schmelzeisen and Schliephake 1998).

The advantages of microvascular free flaps are a secure watertight dural closure, the obliteration of any

dead space, an efficient separation between paranasal sinuses and intracranial space and the protection and support of neural structures and bone grafts (Eufinger et al. 1999; Stepnick 1999).

Flap techniques for skull base reconstruction

Local flaps

- Temporal flap
- Pericranial flap
- Galea flap

Free flaps (fascio-cutaneous)

- Lateral upper arm flap
- Anterior lateral thigh flap
- Rectus abdominis flap
- Scapula/parascapular flap
- Radialis forearm flap
- Latissimus dorsi flap
- DIEP flap

The selection of free flaps is based upon:

- Approach and possibility of integration
- Necessary volume
- Possible anastomoses
- Possibility of contouring

Control of the vitality of the inserted microvascular flaps and skin paddles is important, but difficult. In extreme situations angiographies or endoscopic controls may be necessary (Fig. 13.5).

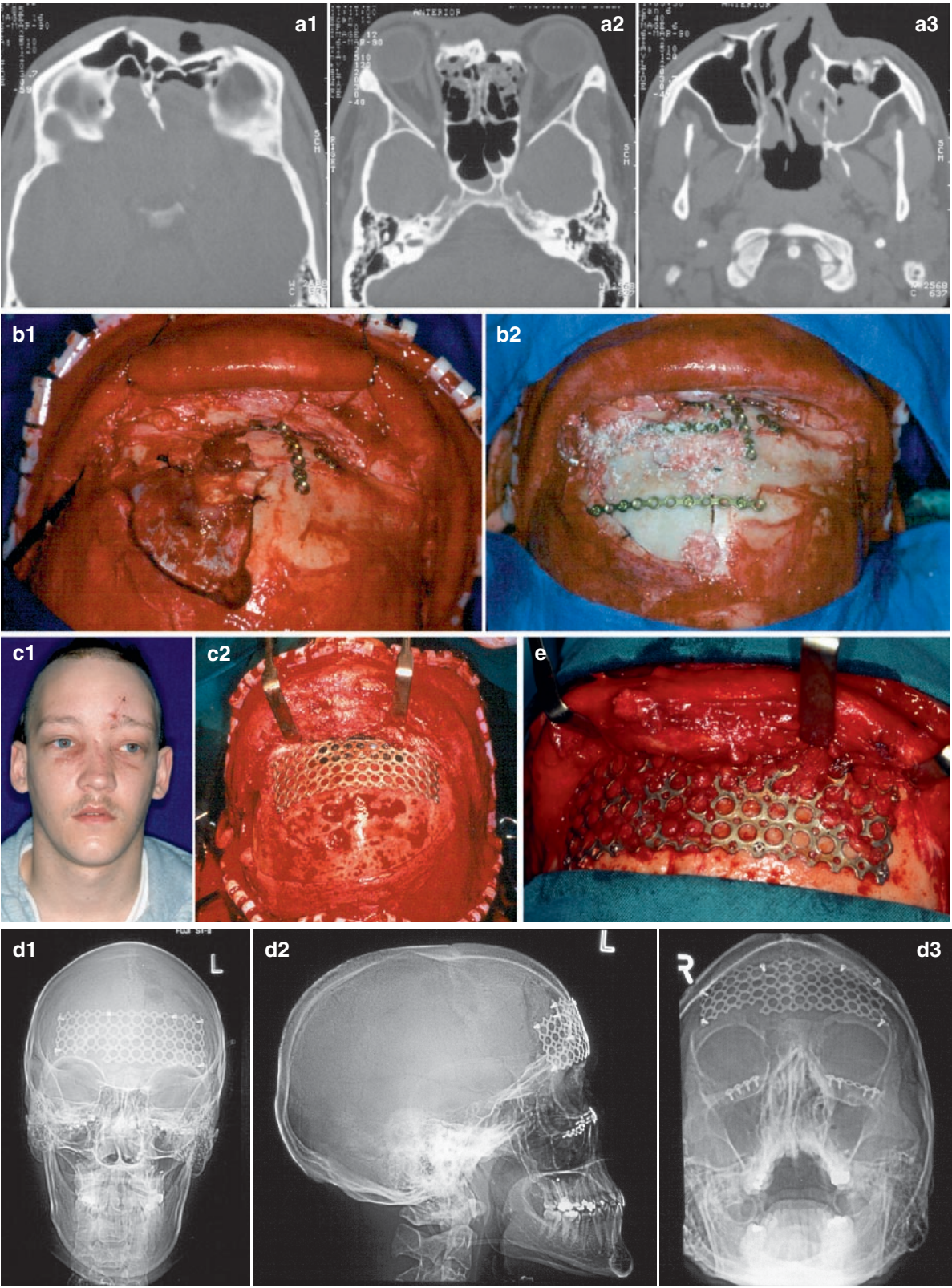
13.4 Hematoma: Central Edema

In 2% of our patients, an epidural hematoma developed in the postoperative phase, which had to be evacuated according to the progression evident on computed tomography (CT). Meticulous intraoperative hemostasis, watertight dural occlusion, tack-up sutures and a subgaleatic suction drainage are important prophylactic measures.

Irreparable cerebral damage from central edema (2%) and severe cranio-cerebral trauma with extensive hemorrhage were responsible for the postoperative mortality in 4% of our patients.

13.5 Subdural Hygroma

Subdural effusions are an accumulation of fluid between the dura and arachnoid membrane. They occur as a result of cranio-cerebral injury and, rarely, as a postoperative complication of craniofacial injuries. Their treatment



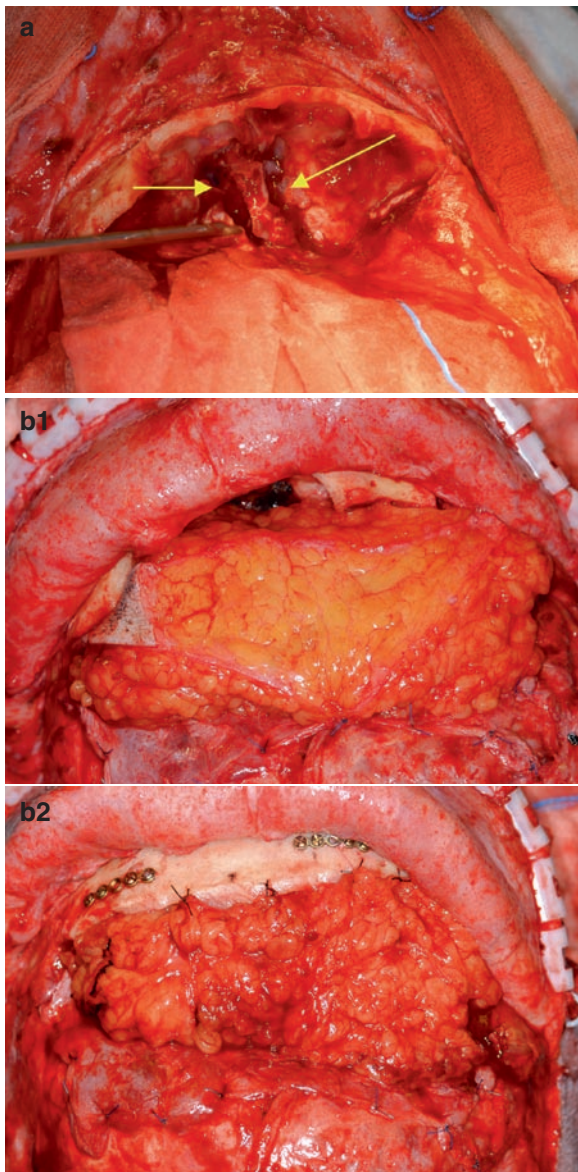


Fig. 13.5 Microvascular free flaps for coverage a traumatic liquor fistula 17 years after severe craniofacial trauma with extensive loss of bone in the anterior skull base. (a) Skull base exposure with large skull base defect (arrow) after bilateral craniotomy and frontal osteotomy. (b) Insertion and anastomosis of a microvascular DIEP flap before and after reintegration of the frontal segment and coverage of the lacerated and scarred dura with a parieto-occipital pericranial flap

corresponds with that of subdural hematomas with recraniotomy and hygroma removal as well as tack-up sutures and placement of an epidural suction drainage (Schmidek and Sweet 1988) (Fig. 13.6).

13.6 Frontal Sinus: Complications

If there is remaining mucosa following cranialization of the frontal sinus, this may lead to formation of mucoceles, pyoceles, and epidural abscesses (Lädrach 2007: 2%). We observed mucocele formation at a frequency of 3.3% in the region of the frontal sinus (Neidhardt 2002).

These patients were re-craniotomized, the mucocele removed and the residual dead space filled either with a pericranial flap or cancellous bone grafts (Baker et al. 2003) (Figs. 13.7, 13.8 and 13.9).

13.7 Functional Neurological Deficits

Nearly 7% of our patients had persisting anesthesia of the supraorbital nerve. In 3%, permanent weakness of the frontal branch of the facial nerve was found, and in 4% there was a traumatic injury of the optic nerve with loss of vision. In 2% of the patients, posttraumatic epilepsy occurred as a result of the primary trauma. Olfactory dysfunction (hyp- or anosmia) as a result of the surgical intervention (transfrontal-intracranial) was found in 8% (Neidhardt 2002).

13.8 Meningitis

Meningitis following skull base revision is always a sign of persisting, mostly occult liquororrhea. Every suspicion of liquororrhea must, therefore, be examined until finally proven otherwise. There was no occurrence of meningitis in our patient group, whereas Lädrach (2007) observed postoperative meningitis in 2.1%.

Fig. 13.4 Frontal osteomyelitis. Reconstruction of the cranio-frontal region with titanium mesh (a1–a3). Depressed cranio-naso-orbito-maxillary fracture. (b1–b2) Primary reconstruction of the frontocranial and naso-ethmoidal region. (c1) Chronic infection with cutaneous fistula formation (5 weeks after primary

reconstruction). (c2) Revision with removal of all infected bone fragments and the osteosynthesis material and reconstruction with titanium mesh. (d1–d3) Postoperative X-ray control. (e) Integrated titanium mesh 12 months postoperatively. No signs of infection

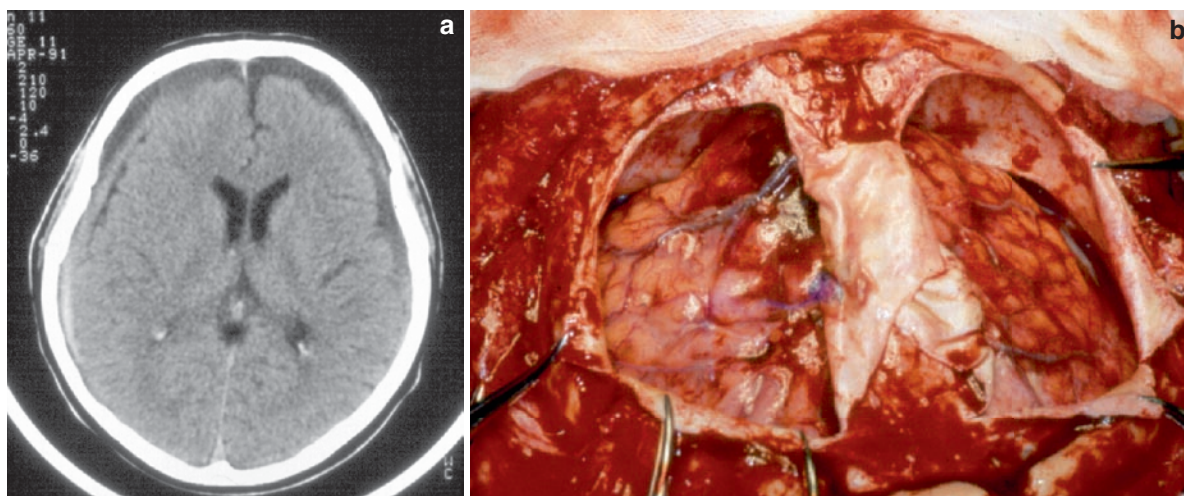


Fig. 13.6 Subdural hygroma following craniofacial reconstruction. (a) CT: enlargement of the subdural space over both frontal lobes with fluid collection of varying density, partly also fresh blood following hemorrhage. (b) Bilateral opening of the dura demonstrating enlargement of the subdural space



Fig. 13.7 Posttraumatic mucocoele of the frontal sinus. (a) Depression of the orbital roof and caudal dislocation of the left globe by the expanding mucocoele. (b) MRI demonstrating the expanding mucocoele in the frontal sinus. Downward displacement of the left globe and atrophy of the orbital roof. The high T1 signal indicates high protein content



Fig. 13.8 Expanding mucocoele following craniofacial treatment (8 years postoperatively). (a) Preoperative caudal dislocation of the left globe. (b) Bone resorption as a result of expansion of the mucocoele. (c) Contour reconstruction with frontal bone fragments following extirpation of the mucocoele. (d) Filling of the defect with cancellous bone grafts. (e) Reconstruction with titanium mesh. (f) Correct postoperative contour normal position of the globe

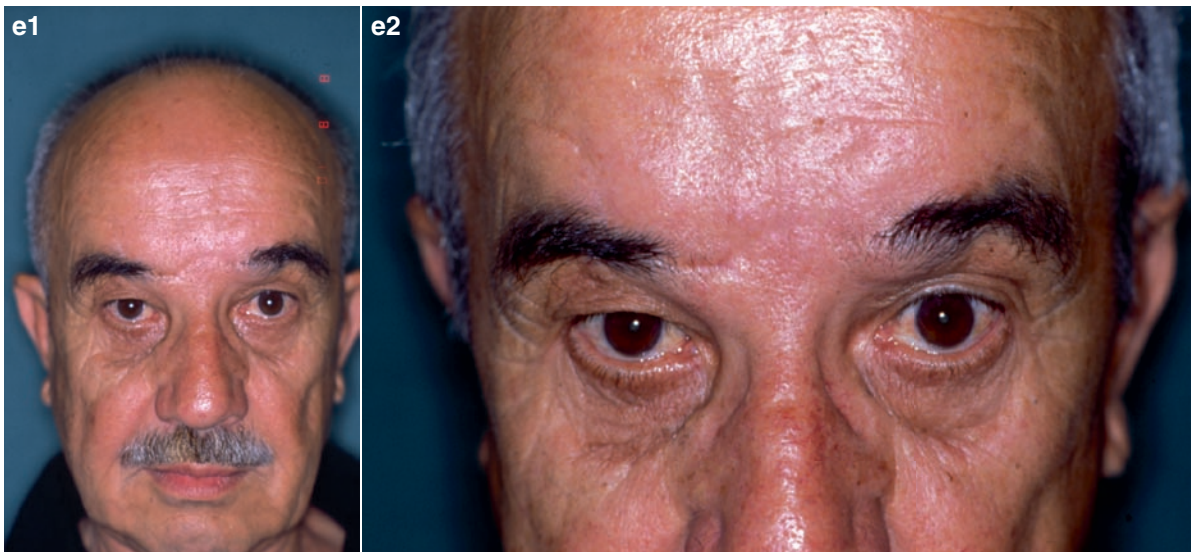


Fig. 13.8 (continued)

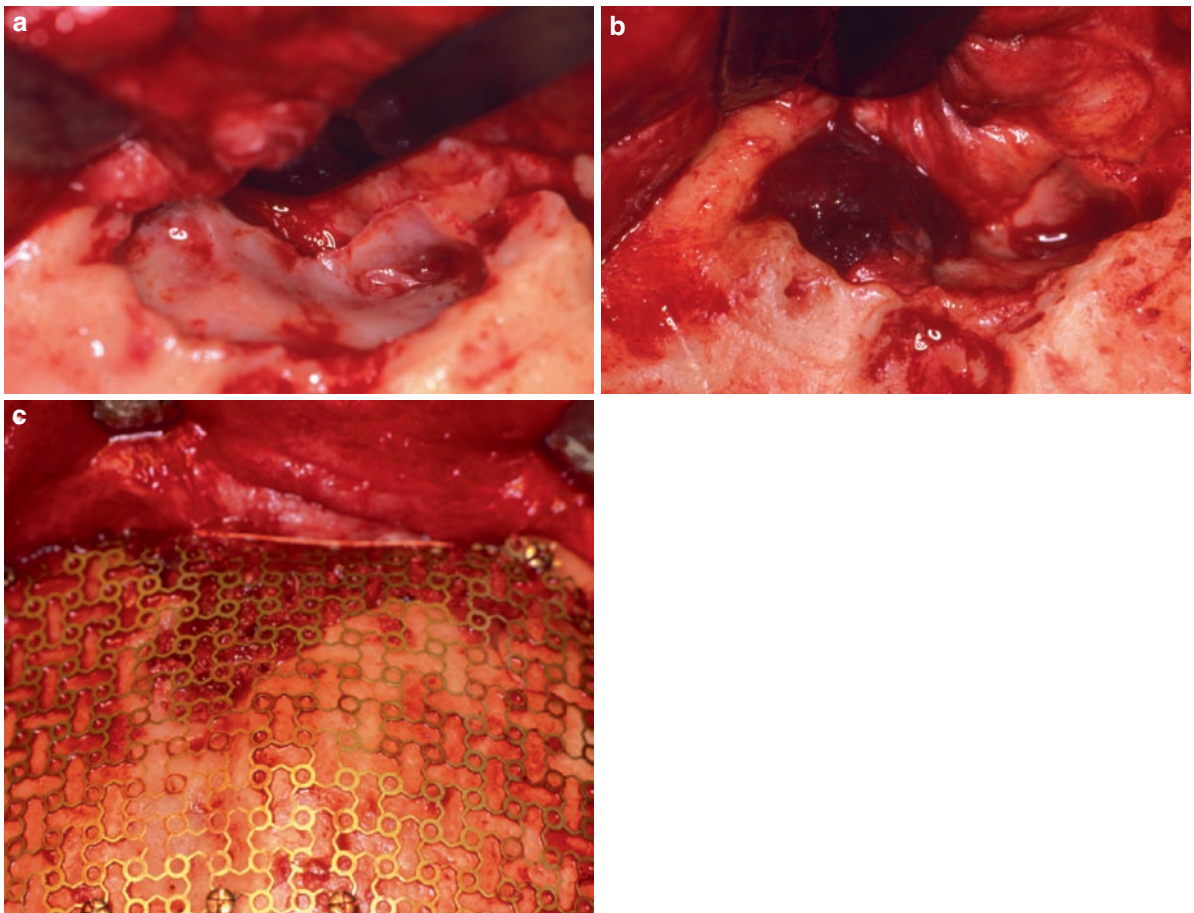


Fig. 13.9 Expanding mucocoele of the frontal sinus. (a) Mucocoele of the frontal sinus. (b) Extirpation of the mucocoele. (c) Sinus obliteration with homogenized cancellous bone grafts and coverage with stabilizing titanium mesh

13.9 Facial Contour Irregularities

Contour irregularities are caused either by the surgical intervention or by bone resorption and have to be corrected due to aesthetic as well as functional reasons (Hardt and Steinhäuser 1979).

Contour irregularities, which required secondary corrections, were found in 4% of our patients.

Minor defects were corrected with bone cement. Major defects were usually treated using calvarial bone grafts or titanium meshes and cancellous bone grafts. In 6% of the patients, scar corrections due to extensive scar formation were necessary (Neidhardt 2002).

13.10 Conclusion

Considering the severity of cranio-cerebral injuries of the 125 patients with complex craniofacial traumas treated in our series the number of posttraumatic functional and aesthetic deficits is relatively low. Early and late complications are not totally avoidable due to the complex anatomical relationships within the craniofacial junction. The surgical techniques (transfrontal-intracranial) presented for treating these injuries help to minimize early complications and late sequelae (Schroth et al. 2004).

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Specific problems arise during the delayed reconstruction of posttraumatic craniofacial defects and deformations, which have to be treated for both aesthetic and functional reasons (Evans et al. 1985; Manson et al. 1986; Merten and Luhr 1994; Kuttenger et al. 1996; Sullivan and Manson 1998).

- An anatomically, functionally, and aesthetically correct reconstruction of the craniofacial structures protects the brain from damage and late injuries and avoids secondary neurological disturbances, infections, and stigmatizing disfiguration.

In our own series, secondary reconstruction was necessary with the following indications:

- Defects not reconstructed during primary treatment, 35%
- Contour irregularities due to bone resorption, 55%
- Contour irregularities due to infection, 10%

14.1 Reconstruction Materials and Techniques

Craniofacial bone defects can be reconstructed using different techniques and implant materials. The choice of the implant material depends on the size and shape of the defect to be reconstructed and on the conditions of the recipient area.

- Reconstruction with autogenous bone/cartilage (calvarium/rib/iliac crest)
- Reconstruction with xenogenous bone/cartilage (bovine/equine bone/lyophilized cartilage)
- Reconstruction with alloplastic bone substitutes (carbonate-calcium-phosphate bone cement, PEEK)
- Reconstruction with titanium-mesh systems

- Reconstruction with preformed titanium implants
- CAD/CAM implants from different materials (titanium/medical raisins)

14.1.1 Autogenous Grafts

14.1.1.1 Split Calvarial Grafts

When reconstructing defects in the cranial skeleton, most authors primarily favor autogenous bone, mainly split calvarial grafts (Lee et al. 1995).

In large defects, autogenous split calvarial grafts provide better results due to their contour stability compared with autogenous rib, iliac crest and deep freeze autogenous calvarial bone grafts (Frohberg and Deathevage 1991; Dufresne et al. 1992; Salyer 1989, 1992; Mathog 1992; Hardt et al. 1994; Lee et al. 1995; Prein 1998; Sullivan and Manson 1998).

Calvarial grafts are usually taken from an area neighboring the defect and separated into tabula externa and interna. Because of their convexity, the grafts (tabula interna) are easily adapted to the frontocranial defect (Dempf et al. 1998).

After integrating the split calvarial grafts, small remaining gaps are filled with bone dust or bone chips previously collected during the craniotomy.

- Calvarial grafts and titanium mesh

An aesthetically efficient symmetrical contour can be achieved by additional titanium mesh coverage in large defects treated with split calvarial grafts. Conspicuous irregularities caused by resorption can simultaneously be avoided. The mesh may be supported by a bone graft to prevent it from sinking in the immediate postoperative phase (Hardt et al. 1994; Kuttenger et al. 1996).

Irregularities in the areas between the grafts and between graft and genuine calvarium can be avoided by additionally covering the contact zones with micro-mesh strips (Hardt et al. 1994) (Figs. 14.1–14.4).

14.1.1.2 Cartilage Grafts

Cartilage grafts can be added, if major contour irregularities of the whole forehead have to be corrected and if graft resorption must be reduced to a minimum to maintain good aesthetic long-term results (Probst 1971, 1973, 1986).

14.1.2 Xenogenous Bone/Allogeneous Cartilage Transplants

Calvarial grafts are usually incorporated as living tissue with reparative and osteoconductive capability (Lee et al. 1995).

This capacity has also been demonstrated for autolyzed antigen-extracted xenogenous bone, such as equine

or bovine bone (Kübler et al. 1998; Tsukagoshi et al. 1998; Eufinger et al. 1999), and lyophilized allogeneous cartilage (Sailer 1983; Sailer and Kolb 1994), which have been used in cranio-maxillo-facial reconstruction for many years.

The complicated processing of these tissues, however, has prevented their widespread use so far.

- Reconstruction of craniofacial bone defects with autogenous or allogeneic bone transplants may lead to minor irregularities caused by bone resorption.

14.1.3 Alloplastic Bone Substitutes (Holmes 1990; Leipziger and Dufresne 1992)

Although autogenous calvarial grafts provide good results, there are nevertheless several limitations:

- Autogenous bone grafts are associated with additional donor-site morbidity

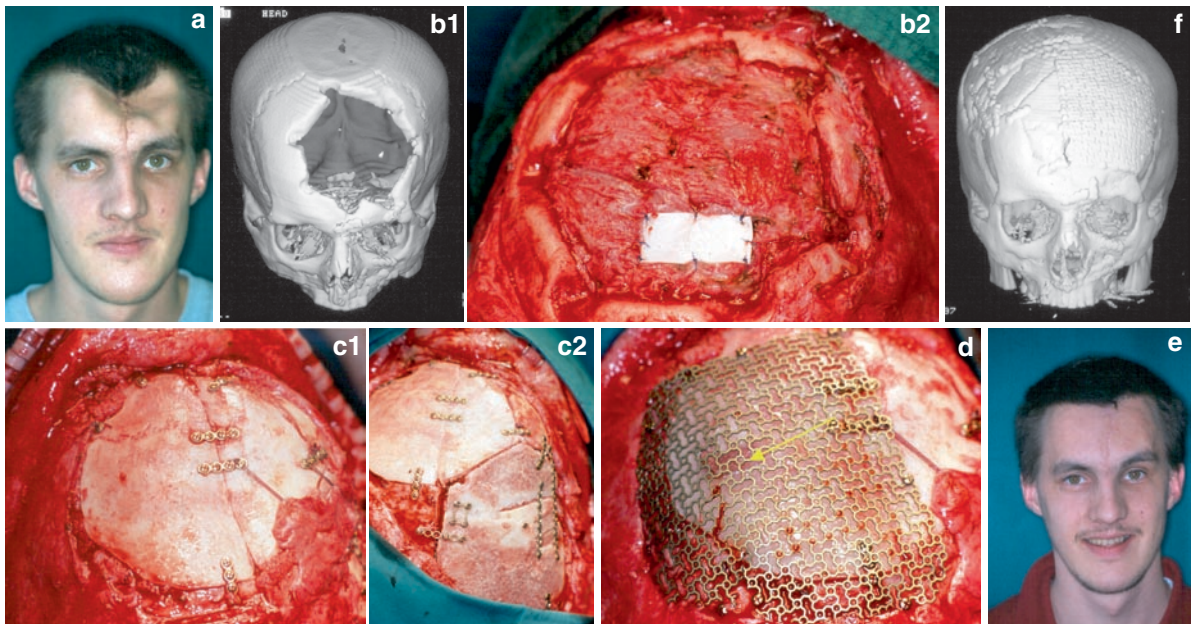


Fig. 14.1 Reconstruction of an extensive fronto-glabellar defect with split calvarial graft and contouring with a titanium mesh. (a) Residual extensive defect of the forehead 6 months after primary treatment of a severe gunshot injury. (b) Three-dimensional CT scan depicting the frontal bone defect. (c) Cranioplasty using autogenous split calvarial grafts from the parietal region. (d)

Residual slight contour irregularities in the frontal area were corrected with a titanium mesh (0.6 mm). The space underneath the mesh was filled with an additional bone graft (arrow). (e) Patient 3 years postoperative with symmetrical and smooth contour of the forehead. (f) Postoperative 3DCT scan demonstrating microplates, bonegrafts, and dynamic mesh in place

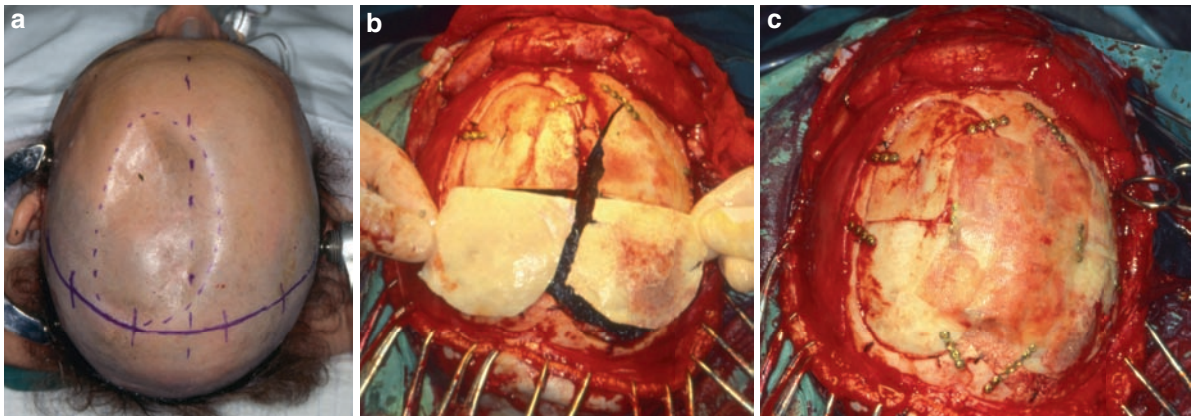


Fig. 14.2 Skull reconstruction after osteoclastic craniotomy. (a) Preoperative depression of the parasagittal parietal skull contour. (b) Secondary bony reconstruction with split calvarial

grafts from the left temporo-parietal region. (c) Completed reconstruction and fixation of the bone grafts with miniplates

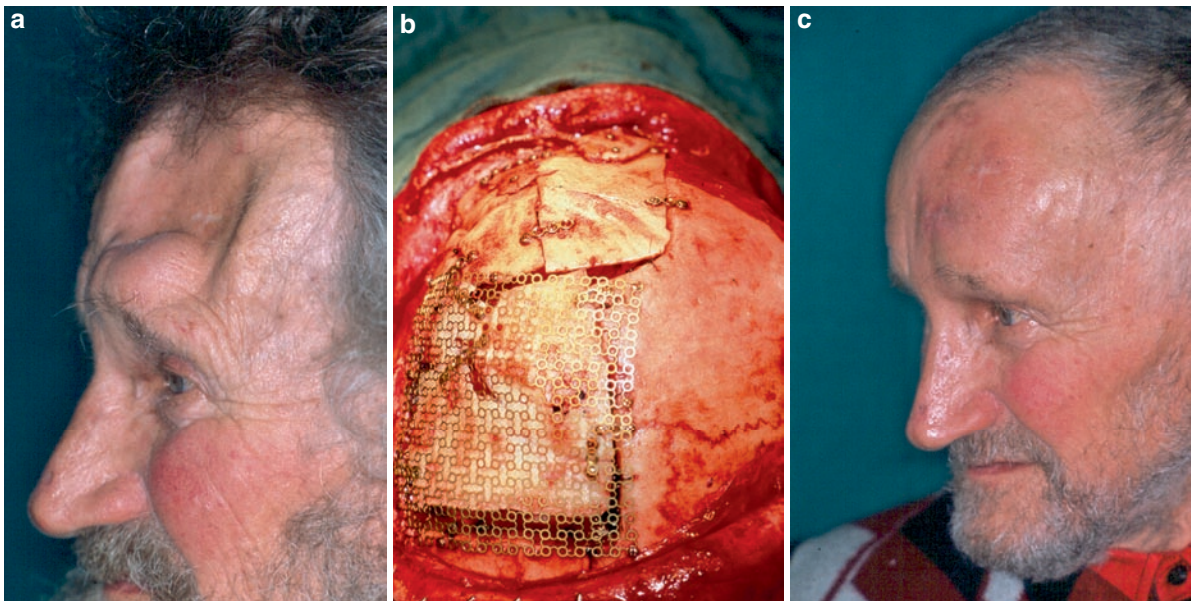


Fig. 14.3 Secondary reconstruction of the forehead with split calvarial grafts and titanium mesh. (a) Preoperative situation showing extensive forehead defect after osteoclastic intervention. (b) Intraoperative forehead reconstruction with split calva-

rial grafts from the parietal region. The donor region is covered with a titanium mesh (0.3 mm). (c) Postoperative result showing smooth and symmetrical contour of the forehead

- Adequate form and sufficient quantity are not always available
- Unpredictable resorption may occur

As a consequence, various alternative substitutes are used in craniofacial surgery, which do not require a second donor site and guarantee an unlimited availability of noninfectious material (Holmes 1990).

Requirements for alloplastic bone substitutes

- Mechanical long-term stability
- Biointegration/biocompatibility
- Moulding ability
- Contour stability
- Favorable cost-factor

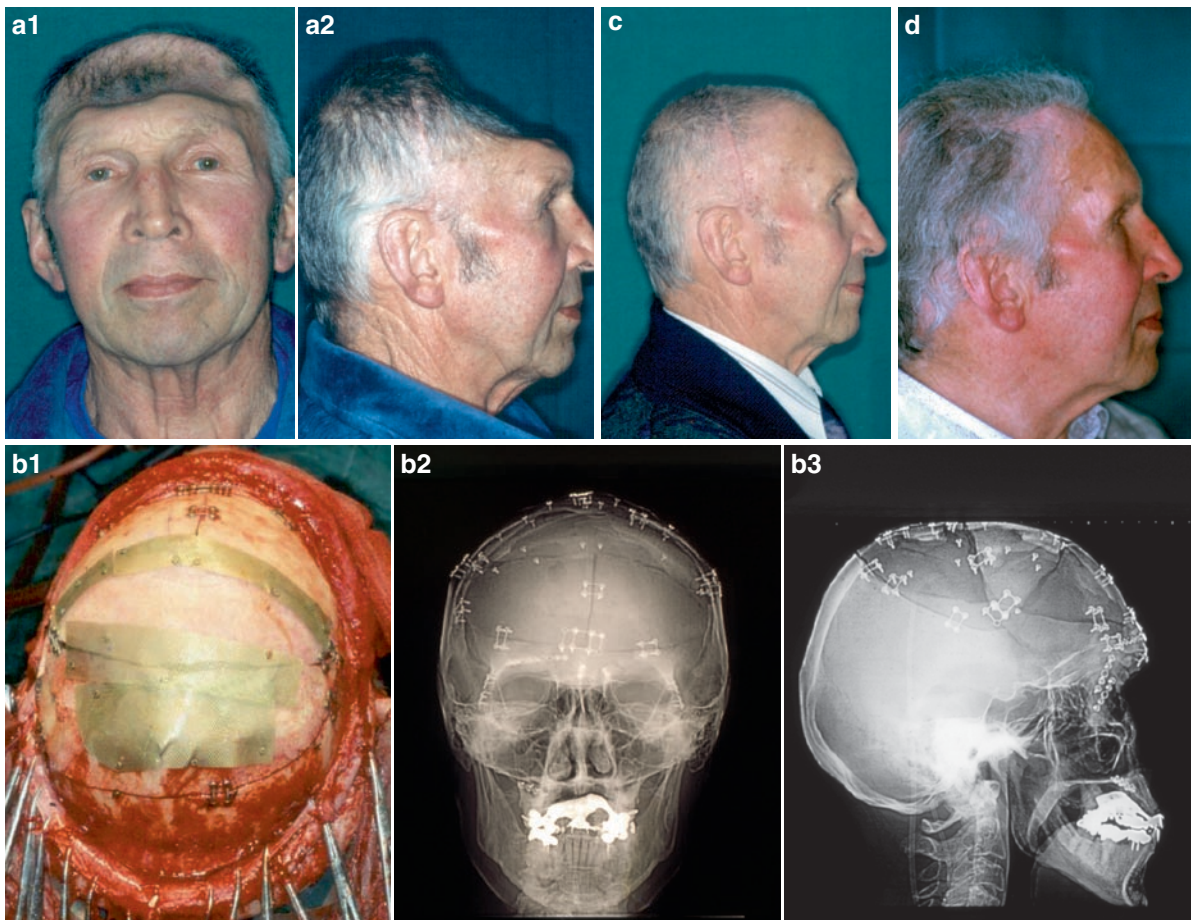


Fig. 14.4 Secondary forehead reconstruction. (a) Extensive fronto-parietal bone defect. (b) Reconstruction with biparietal split skull grafts. The residual donor defect was closed with an additional iliac

bone graft. Contouring with micro-titanium mesh strips. (c) Postoperative result after 6 months. (d) Postoperative result after 10 years, demonstrating stability of the reconstruction

14.1.3.1 Synthetic Calcium Phosphates

These substitutes vary greatly in their properties (osteoconductivity, biocompatibility, mechanical stability), as well as having diverse forms of preparation. They are divided into two groups, granulate and cements, which are mixed during surgery and applied as a paste (Holmes 1990; Costantino et al. 1993; Costantino and Friedman 1994).

- Hydroxyapatite granulate

Good results were achieved using hydroxyapatite for cranial reconstruction (Costantino et al. 1991, 1992; Nakayima et al. 1995; Burstein et al. 1997, 1999; Pistner et al. 1998; Byrd et al. 1993; Wiltfang et al.

2004). Pistner et al. (1998) pointed out that a dry operating field is mandatory when using hydroxyapatite. This is certainly not always possible in craniofacial surgery.

- Bone cements

In general, the modern carbonate-calcium-phosphate bone cements exhibit good biocompatibility, which is based upon their osteoconductive properties, unhindered biodegradation and osteoclastic resorption with osseous replacement (Constantz et al. 1995; Frankenburg et al. 1998; Smart et al. 2005).

In addition, there is a direct bone apposition on the surface of the bone cement without connective tissue interposition even in the early postoperative phase

(Constantz et al. 1995; Jupiter et al. 1997; Frankenburg et al. 1998).

Once the bone cement has set and healed, restoration is practically identical to that of autogenous bone grafts. Clinically and aesthetically stable results can be achieved either alone or in combination with split calvarial grafts (Wiltfang et al. 2004).

- Clinical indications (Mahr et al. 2000; Baker et al. 2002; Kirschner et al. 2002; Losee et al. 2003; Wolff et al. 2004)
 - Filling craniotomy defects and craniotomy holes or resorptive and untreated traumatic defects.
 - Augmentation of traumatic and nontraumatic defects or irregularities in aesthetically demanding regions, such as in the periorbital (supraorbital margin) and zygomatico-facial region.
 - Reconstruction of the osseous skull base.
- Surgical techniques

After exposing the defects and preferably trimming the margins vertically, cements can easily be applied and formed. The bone margins provide sufficient stability. Maximum rigidity on compression (30 MAP) is achieved approximately 24 h after application and is equivalent to two- to six-times that of cancellous bone (Figs. 14.5 and 14.6).

14.1.3.2 Synthetic Polymers

Synthetic polymers are not reabsorbable; there is no possibility of remodeling and they may lead to inflammatory tissue reactions due to thermic and toxic reactions.

Even after a number of years tissue damage can be observed, which makes their application controversial.

Polymethyl-methacrylate (PMMA-palacos) has been used extensively in cranioplasty (White et al. 1970; Cabanela et al. 1972).

However, a complication rate of 2–12% within the first 2 years was reported (Cabanela et al. 1972; Henry et al. 1976). Nowadays, PMMA may be coated with bone marrow-impregnated (poly-DL-lactic-co-glycolic acid) foam to improve osseointegration in the cranioplasty (Dean et al. 1999).

New medical PMMA products are under development and may be ready for use in the near future.

Polyetheretherketone (PEEK) is another material of interest for the reconstruction of calvarial bone defects. However, its use is at present limited to extended defects with reconstructions based on computer-aided design and manufacturing (see Chap. 15).

14.1.4 Titanium-Mesh

Titanium mesh systems can be used for primary reconstruction in nonload-bearing areas. In secondary reconstruction, these meshes can be combined with autogenous bone grafts (Esser and May 1990; Hardt et al. 1994; Kuttenger et al. 1996; Kuttenger and Hardt 2001).

Irregularities between bone grafts and in the contact zone between graft and genuine calvarium can be avoided by covering the gaps with titanium micromesh strips (Hardt et al. 1994).

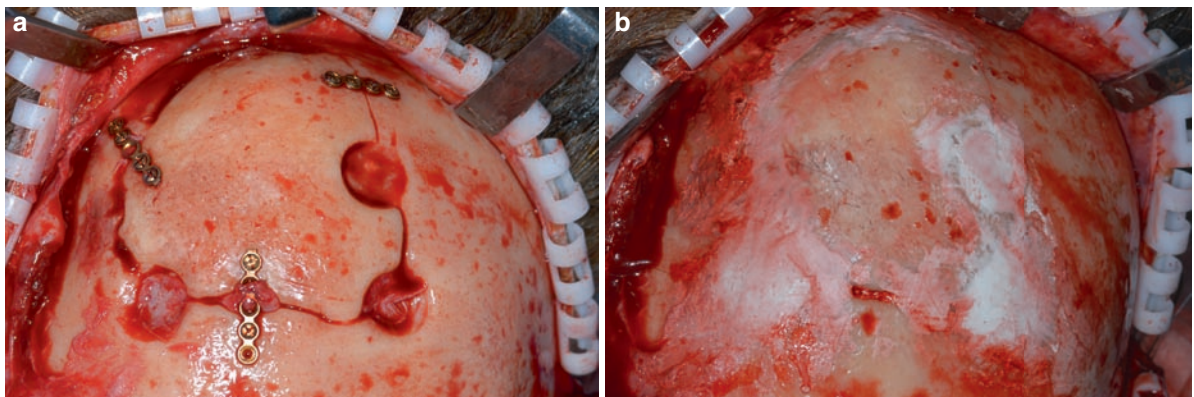


Fig. 14.5 Cranioplasty with bone cement (NORIAN-CRS cement). (a) Frontal contour irregularities after previous craniofacial trauma and craniotomy. (b) Residual bony defects of the drilling holes are filled with bone cement

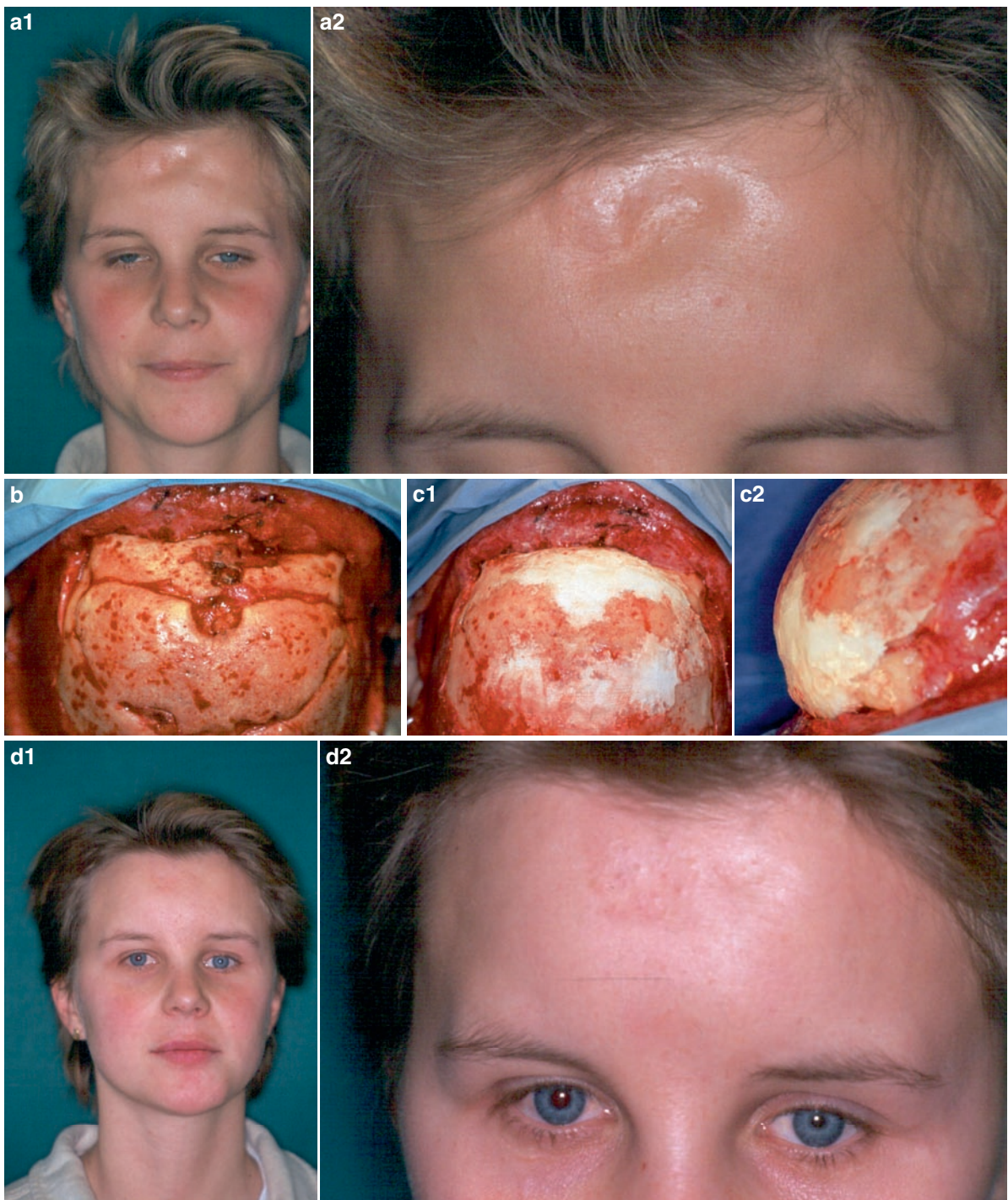


Fig. 14.6 Cranioplasty with bone cement (NORIAN-CRS cement). **(a)** Unaesthetic frontal contour irregularities following previous craniofacial trauma and craniotomy. **(b)** Remaining

frontal osseous defects. **(c)** Intraoperative defect leveled with bone cement (NORIAN-CRS cement). **(d)** Postoperative result showing smooth contour of the forehead

Contour irregularities after removal of infected bone fragments or grafts may be reconstructed using titanium meshes in combination with autogenous bone grafts. Even in a chronically infected situation, undisturbed healing can usually be expected.

Titanium mesh contact with the paranasal sinuses does not pose a problem.

Minor contour irregularities after reconstruction with titanium mesh may appear along the margins of the mesh. These problems can be avoided by correct bending, adaptation, and fixation of the mesh.

In case of secondary displacement and marginal irregularities, the mesh can easily be removed (Figs. 14.7).

- Own results: Titanium mesh

During the follow-up of our patients treated with titanium mesh systems, no mesh-related complications were observed. Neither wound infections, exposures of the mesh nor mesh loss were noted.

In all cases of paranasal sinus wall reconstruction, complete re-pneumatization of the sinus took place.

During the long-term follow-up, all forehead reconstructions exhibited excellent contour stability. Minor irregularities were observed in one patient with extensive panfacial and anterior skull base fractures caused by visible miniplates and screw-heads in the forehead, which had to be removed (Kuttenberger and Hardt 2001).

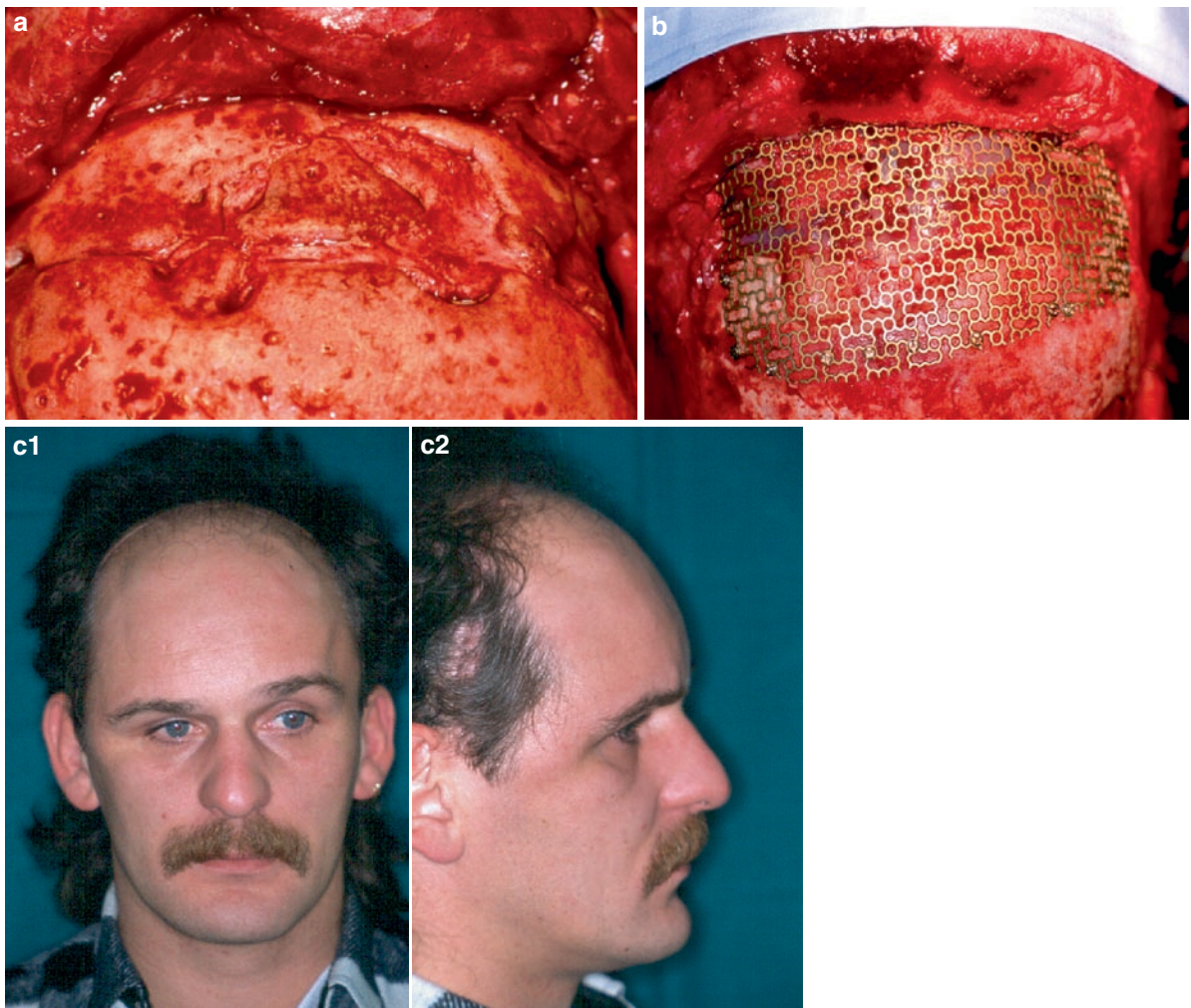


Fig. 14.7 Secondary reconstruction of the frontal region. (a) Intraoperative extensive resorption of the frontal bone after previous craniofacial reconstruction. (b) Intraoperative defect fill-

ing and contouring with autogenous cancellous bone grafts stabilized with a titanium mesh (0.3 mm). (c) Postoperative result with symmetrical and smooth contour of the forehead

14.1.5 Preformed Titanium Implants (CAD/CAM Implants)

Computer-assisted and -fabricated implants (CAD/CAM) offer an alternative to conventional reconstructions. The use of titanium permits the production of fine, but nevertheless stable implants (Wehmüller et al. 1995; Heissler et al. 1998; Eufinger et al. 1995, 1998).

C T-based reconstruction of neurocranial and frontofacial defects with pre-formed titanium implants avoids additional functional problems, reduces the risk of infection in comparison with other procedures and provides a precise and individual fit (Naßberg 1995; Eufinger et al. 1998). More details about standard procedures and new developments are presented in Chap. 15.

Advantages of CAD/CAM implants

- Preoperative, exact virtual three-dimensional planning
- Use of biocompatible materials
- No donor site morbidity
- Good aesthetic results
- High functional protection
- Precise implant fit
- Reliable reconstruction results
- Reduced operating time

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15.1 Overall Objective

The reconstruction of craniofacial defects is a challenging task even for the experienced surgeon. For an optimal solution the techniques and principles that have been described in the chapters before must be adapted to the individual situation. The challenges result from the patient's individual situation, the preoperative planning efforts, from technical aspects as well as from the reconstructive means available. While the use of autografts has been the most widely recommended method, it does have its drawbacks, including long operation times, donor site morbidity, limited donor bone supply, as well as different anatomic and structural problems. The availability of autogenous bone grafts resembling the form of the skull is limited. Therefore, there is a need for alternative materials with adequate mechanical properties and biocompatibility (Blake et al. 1990; Eufinger et al. 1995; Eufinger and Wehmöller 1998, 2002; Klongnoi et al. 2006; Wiltfang et al. 2002, 2003; Schiller et al. 2004; Thorwarth et al. 2005; von Wilmowsky et al. 2008).

15.2 Patient-Related Conditions

The size of the defect, delayed, or inadequate debridement of the wound, delayed, or inadequate treatment of the patient or the relatively high risk of infections are the typical causes for complications in craniofacial injuries. In addition to primary or secondary treatment

attempts, the morbidity of the patient and the risk of the planned reconstructive procedure may result in severe complications. Concerning the patient situation, the following questions have to be answered before an attempt for reconstruction is made:

- Size and location of the defect
- General health status
- Neurological status
- Patient's wish/compliance
- Treatment plan
- Technical aspects

15.2.1 Size and Location of the Defect

The size of the defect plays an important role in the determination of the reconstructive procedure. Poukens et al. (2008) have developed a classification as a basis for the intended reconstruction. When designing skull implants, the sometimes complicated geometry of the defects is important to consider. Defects crossing the midline, or defects including the orbital rim or roof of the orbit, pose a greater challenge for design and manufacturing than a simple one-sided parietal bone defect. Apart from the size and extent of the defect, these anatomical factors ought to be included when classifying skull defects. The proposed classification (Table 15.1) is, therefore, based on three factors that determine the complexity of the reconstruction:

- Size of defect in cm²
- Defects crossing the midline of the skull
- Involvement of the orbital rim/roof

The size of skull defects is important for obvious reasons – the larger the defect, the larger the span of curvature that has to be reconstructed. The implant should

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Table 15.1 Classification of cranial implants based on the degree of difficulty in computer design and manufacturing

	<5 cm ²	5–100 cm ²	>100 cm ²	<5 cm ² + orbital involvement	5–100 cm ² + orbital involvement	>100 cm ² + orbital involvement
No midline crossing	I	II	III	IV	IV	V
Midline crossing	I	II	III	IV	IV	VI

be designed in a way that the contour of the surrounding skull bone is followed. Small defects of <5 cm² have an almost flat surface, whereas large defects require a more sophisticated implant, which has implications on the design procedure.

Defects that cross the midline pose more problems when designing the implant, since information on the contour of the contralateral side of the skull is missing and, thus, mirroring of the contralateral curvature is not possible. Designing an implant that involves part of the orbit is more complicated because of the curvature of the orbital area and the need for mirroring of the other side. The anatomical location of the defect is, of course, also important, since the bone thickness varies along the skull. The thickness of the bone has to be taken into account when designing the implant, especially the part that will be fixed to the skull bone. The proposed classification divides skull defects into six distinct classes, based on the difficulty of implant design. A skull defect is described by the class it belongs to and its anatomical location.

15.2.1.1 Examples

Patient 1 had undergone previous surgery for removal of a meningioma, with creation of a defect at the left parieto-occipital area of the skull (Fig. 15.1a–d). The skull defect was classified as a Class II occipital skull defect. Design and manufacturing was based on a multispiral computer tomography (CT) scan (Toshiba, Japan). The scan data were converted into a stereolithographic (STL) data set using the computer program Mimics (Materialise, Leuven, Belgium). The STL data were imported in 3Matic (Materialise, Leuven, Belgium) and further processed on a Pentium IV workstation. It was decided to manufacture a titanium implant, using high-speed milling (HSM).

Patient 2 had undergone surgery for decompression of the brain after severe trauma. A part of the skull bone became necrotic, leaving a large skull defect at the right

side (Fig. 15.2). The skull defect was classified as a Class III temporo-parietal skull defect. The computer-aided design resembled the procedure in case 1. The implant, however, was manufactured by selective laser melting with titanium powder (SLM).

The implant in patient 1 was designed in 3 h with the help of surface guidelines and mirroring of the contralateral side (Fig. 15.1b) (Hutmacher et al. 2004). Four fixation lips on the implant were designed to allow fixation to the skull. The implant (Fig. 15.1c) was milled out of medical grade 5 titanium block in a five-axis HSM machine in 48 h (IDEE, MUMC Maastricht University Medical Centre, Maastricht, The Netherlands). The implant was fixed with four titanium screws (length 3 mm, diameter 1.5 mm, KLS Martin, Tuttlingen, Germany) to the remaining skull (Fig. 15.1d).

The implant of patient 2 was designed in 16 h with the help of surface guidelines and mirroring of the contralateral side. Five fixation lips on the implant were designed to allow fixation to the skull. The implant (Fig. 15.2b, c) was manufactured out of medical grade 5 titanium alloy by Electron Beam Melting (Arcam, Sweden) in 12 h. The implant was fixed with five titanium screws (length 3 mm, diameter 1.5 mm; KLS Martin, Tuttlingen, Germany) to the remaining skull (Fig. 15.2d).

15.2.2 General Health Status

The medical history of the patient as well as size and localization of the defect are crucial elements for the determination of the treatment plan. The anamnesis must consider the exact medical history of the patient, as different defect causes — trauma, tumor, infection, neurological diseases, apoplectic insults, malformation — may lead to a different evaluation and treatment plan.

Coexisting medical problems may even prevent any further attempt at reconstruction:

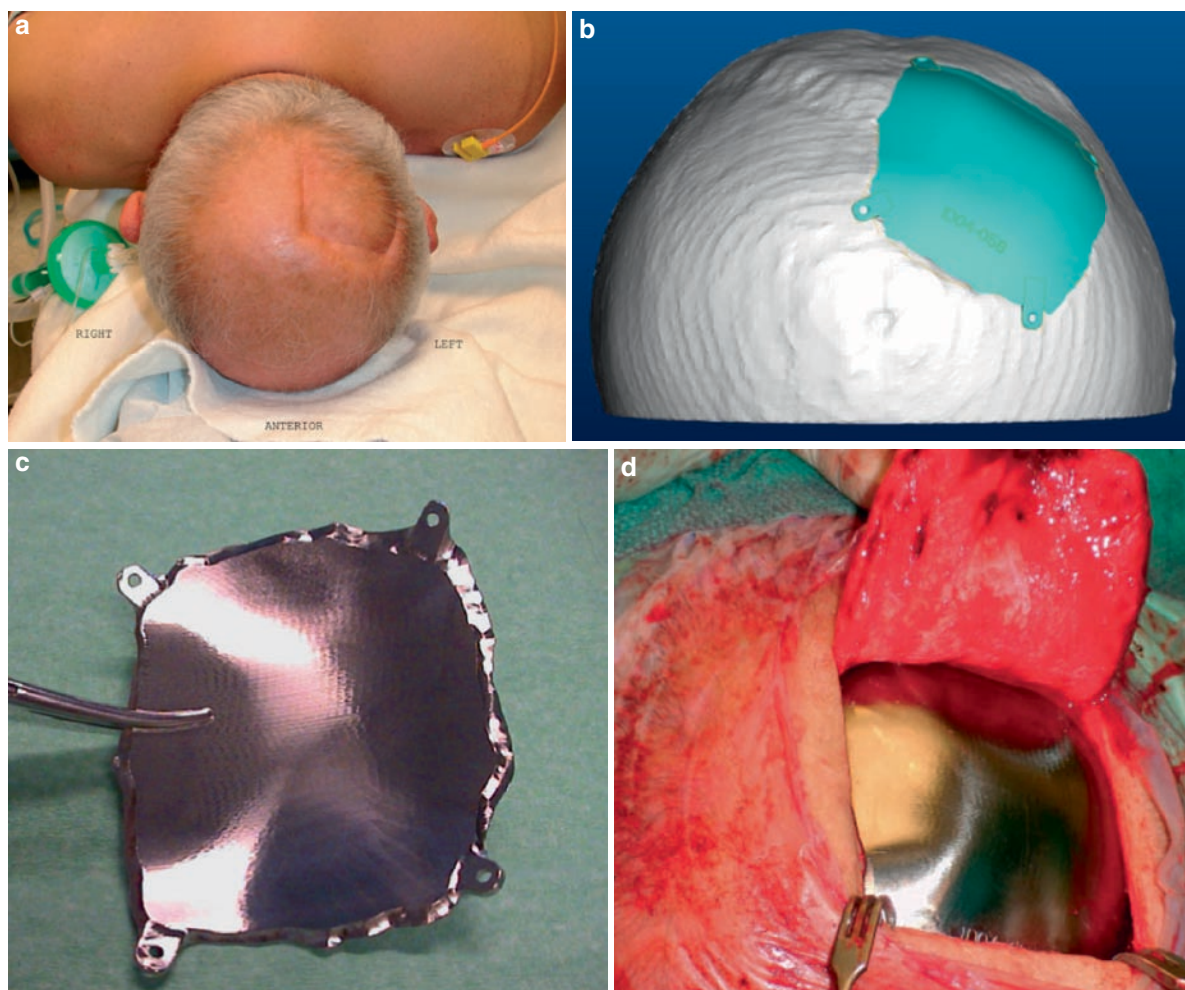


Fig. 15.1 (a) Impression at the left parieto-occipital area from previous surgery for removal of a meningioma. (b) Rapid prototyping model shows the large skull defect. (c) Implant designed for the left parieto-occipital defect. (d) Implant placed in the defect

- Age, life expectancy and prognosis
- Severe medical disorders
- Multiple treatment failures due to infections or others
- Anticoagulative medical treatment
- Deficits in cooperation
- Alcohol or drug abuse

The duration of the operation plays a considerable role in the reduction of risks. It reduces the blood loss and the risk of brain swelling, the infection risk and the risk of tissue damage. The indirect reconstruction of defects using prefabricated allogenic implants reduce the operation time, whereas a direct reconstruction with autogenous bone material causes higher morbidity and is more time-intensive due to harvesting the bone specimen.

There are, however, patients where no further attempt at reconstruction should be considered. These are patients with a clearly reduced life expectancy or patients that do not accept any reconstruction. Drug or alcohol abuse may be further contraindications for reconstruction. In patients suffering from apoplectic insults or intracranial bleeding with a high risk of thrombosis or embolism, the surgical risk and the method of reconstruction must be calculated with respect to the anticoagulative therapy. In this group of patients, allogenic implants are advantageous as the surgical intervention should be limited to the defect reconstruction. A limited surgical intervention with an acceptable duration of the operation will help to reduce bleeding to a minimum. Nevertheless, the risk of thrombembolism must be respected and evaluated as a surgical intervention is planned. A preventive low-dose

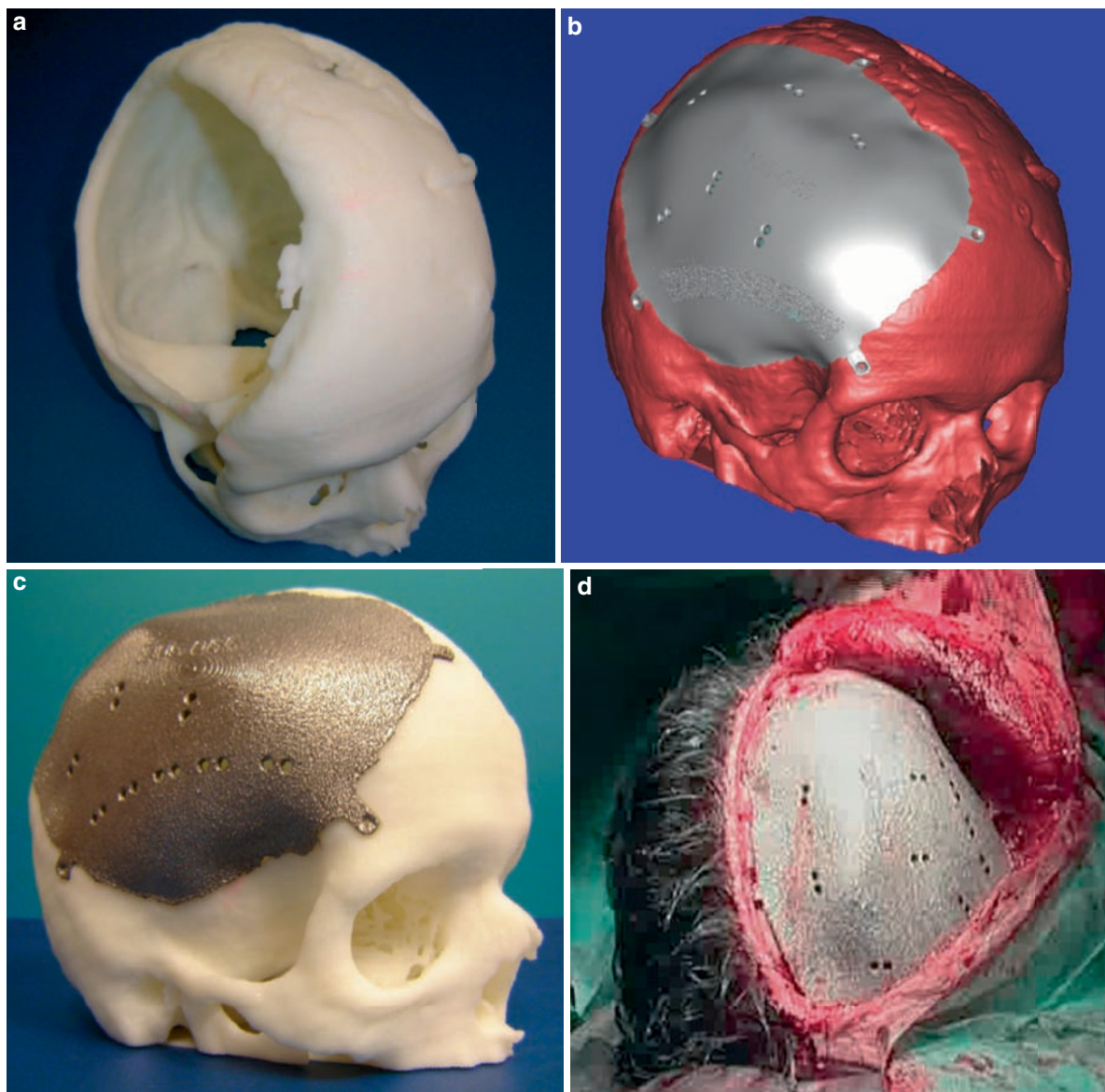


Fig. 15.2 (a) Rapid prototyping model shows the large parieto-fronto-temporal skull defect. (b) Implant designed for the reconstruction of the defect. (c) Selective laser melting (SLM)-made implant. (d) SLM-made implant placed in the defect

medication with NSARs should not be stopped. A second intervention (e.g., the harvesting of autogenous bone material) should be avoided, however.

Small defects (class I) as the result of skull bone trepanation or trauma, for example, may not always be reconstructed. The decision depends on the localization of the defect, the age of the patient and the risk of injury in the affected region. In young, non-handicapped, active patients there may be a higher need to reconstruct defects as they may limit an individual's freedom and quality of life. Also, aesthetic

aspects — especially in the forehead region — are considered as a serious indication for reconstruction. In older patients with high comorbidity risks due to medication or age, the necessity to reconstruct skull bone defects may be evaluated differently, as the possible improvement must be seen in relation to the general risk of the operation.

There is also a small group of patients where other solutions should be considered than the methods mentioned above. These are patients, where multiple attempts using different techniques of reconstruction have failed.

As soft tissue coverage is essential for success with allogenic and autogenous materials, it may in some rare cases not be advisable to reapproach these patients again with a surgical solution. For these patients, helmets and epithetic applications help to reduce the risk of injury and improve the aesthetic appearance.

15.2.3 Neurological Status

Neurological deficits may be the consequence of skull bone trauma, intracranial tumors or apoplectic insults. Besides the general medical status of the patient the necessity, urgency, and method of skull bone reconstruction depend on the patient's neurological status. The neurological deficit can result from the disease leading to the skull bone defect, but also from the defect itself. To detect and evaluate the skull bone defect as primary or additional cause for a neurological deficit, however, is difficult. Nevertheless there is a need for reconstruction of skull bone defects to improve the individual's neurological situation and protect the brain from further damage. The neurological deficits can be temporary, long lasting or even progressive. Traumatic brain injuries may heal without consequences, but in 35% of the cases with open brain traumas traumatic epilepsy develops (Poeck 1987). Nearly 95% of these traumatic epilepsies develop in the first two posttraumatic years (Poeck 1987).

The most obvious indication for skull bone defect reconstruction is to prevent further damage to the patient. Sensory or motor deficits, hemi-, or paraplegic paralysis, epileptic attacks or infections will lead to a progressive loss of motor and sensory function and control. Medical treatment and rehabilitation will result in a sensory-motor improvement or stabilization in many patients.

The better the functional results are, the more urgent is the need for a skull bone reconstruction, especially in large bone defects. This implies that the reconstruction of skull bone defects is necessary to avoid any further traumatic brain damage resulting from a direct trauma to the brain (Poeck 1987).

In patients with long-persisting neurological deficits the rehabilitation efforts may be supported by the reconstruction of skull bone defects in two ways:

1. The integrity of the skull lowers the risk of a further injury
2. The skull bone reconstruction itself may improve the neurological status

Aesthetic rehabilitation due to skull form may be another indication for reconstruction, especially in patients with no or few neurological deficits (Blake et al. 1990; Eufinger and Wehmöller 1998, 2002; Poukens et al. 2008).

15.2.4 Patient's Wish

Suboptimal outcomes are not as uncommon as they should be. Careful assessment and analysis of the existing treatment deficit including three-dimensional CT scans and soft tissue evaluation, as well as careful follow-up and audit will establish their full extent. Inadequately treated craniofacial defects can result in significant cosmetic deformity and functional disabilities, which are extremely difficult to correct. If possible, a simulation of the intended reconstruction should be performed to compare different treatment options.

To re-establish a maximum of quality-of-life, function, social acceptance and self-esteem, every reasonable effort should be undertaken to correct and improve a patient's situation and individual cranio-maxillofacial defect. In some cases, a craniofacial implant must be inserted as a prerequisite for further epithetic reconstruction as shown below.

A serious evaluation and discussion with the patient may in some cases lead to the conclusion that no reconstruction should be attempted. This should be considered, if it is the patient's wish and the technical possibilities cannot be harmonized to reach a treatment goal which is acceptable to the patient (Fig. 15.3).

15.2.5 Treatment Plan

A team of specialists has to be at hand to perform the planning, designing, and manufacturing of the implant and reconstruction of the defect. As each step of the procedure has to be validated, a close cooperation between the partners is an absolute necessity for a good result.

The ideal team consists of:

- Cranio-maxillofacial reconstructive surgeon/neurosurgeon
- Radiologist
- Engineering team for design and preparation of data sets
- Engineering team for manufacturing of the implant



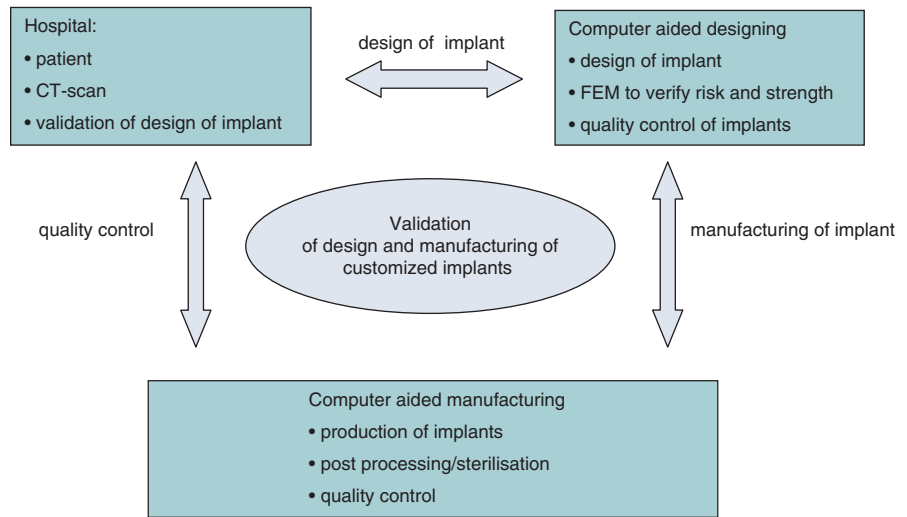
Fig. 15.3 (a) Rapid prototyping model shows the large craniofacial bone defect. (b) Implant for the reconstruction of the latero-orbital defect. (c) Control X-rays after implantation. (d)

Patient before and after reconstruction with titanium implant and epithetic reconstruction of the orbit

- Sterilization unit
- OP capacity with experienced anesthetist and nursing staff
- Intensive care unit

In an ideal setting, the cranio-maxillo-facial surgeon and neurosurgeon work together as a team to perform the reconstructive surgery. The team leader is responsible for the data acquisition, product planning and production of the implant. The leading reconstructive surgeon has to validate the production process step by step, as the engineering teams usually have no direct contact

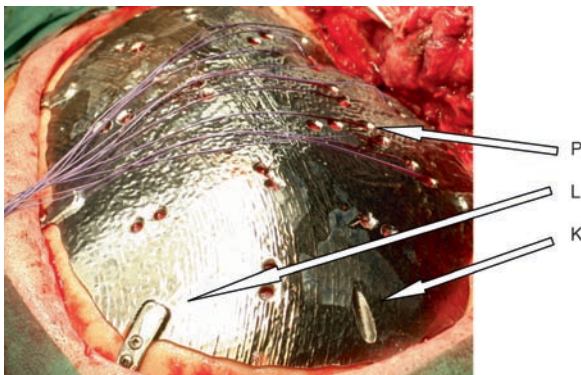
with the patients. He has to organize the whole procedure with respect to the operation date. The production process, the material used, the methods of manufacturing, sterilization of the implant and the transport of the sterilized implant to the operation room must follow a certified pathway. It must be guaranteed that each step of the production process is documented according to internationally accepted quality standards. The whole process from data acquisition to the operation may take at least 6 weeks. Figure 15.4 shows a diagram of the treatment plan.

Fig. 15.4 Treatment plan

15.2.6 Technical Aspects

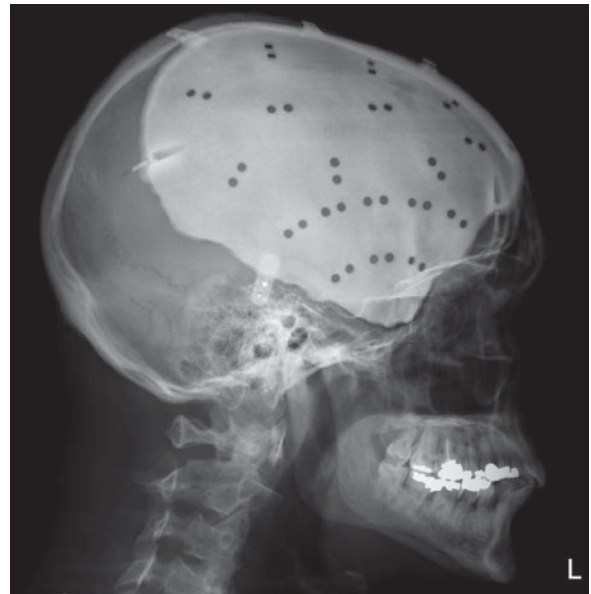
The individually made implant should be fixed with standard titanium screws of 2.0- or 2.3-mm diameter and a variable length. The designing engineer has to consider fixation elements, such as lips or screw canals, for the fixation of the implant. Both elements can be combined.

The fixation lips have to be long enough and may host two drill holes. The reconstructive surgeon has to discuss the design of the implant and the position and number of fixation elements with the engineers. The bicortical layer of skull bone is ideal for a monocortical implant fixation,

**Fig. 15.5** Milled titanium implant. *L* fixation applications, *K* screw canals tangentially designed for direct screw fixation, *P* perforations for tack-up sutures

whereas the region of thin bone from the infratemporal region should be avoided (Figs. 15.5, 15.6).

The patient's head has to be fixed in a Mayfield clamp for an absolute stable fixation. The surgical treatment plan has to be discussed with the responsible anesthesiologist to control and lower the blood pressure to a reasonable level to avoid an unnecessary

**Fig. 15.6** X-ray control after skull reconstruction with the titanium implant displayed above. Absolute exact fit. Fixation applications are clearly visible

blood loss. After the operation, the patient should be transferred to the intensive care unit.

15.3 New Developments

15.3.1 Titanium Selective Laser Melting (Ti-SLM)

Since the mid 1990s allogenic implants were applied to restore skeletal defects in the cranio-maxillofacial region with varying success. Due to incorrect planning, manufacturing, or application, these implants found limited acceptance.

Today, the higher life expectancy and mobility of patients, as well as an increasing number of younger patients in need of such implants, require better solutions than in the past. The highly sophisticated data acquisition and better computer programs have improved the precision and acceptance of the implants.

An improved integration of titanium implants at the titanium-bone interface could be realized by inducing better bone ingrowths by producing a more porous surface structure. The positive effects of increasing the surface area are well known from dental implants (Hattar et al. 2005; Klein et al. 1994; Li et al. 2005). The manufacture of implants with graded mechanical properties concerning reduced weight, full freedom of form and stiffness is not possible with the current state-of-the-art technology.

15.3.1.1 The SLM process

By SLM it has become possible to produce complex, three-dimensional titanium implants directly out of serial materials. Titanium powder is brought onto a work platform in layers of 0.03–0.1 mm thickness. According to three-dimensional computer data sets, a layered implant construction is possible. A focused laser beam of high intensity delivers the energy to melt the powder particles to form a solid titanium implant (Hon and Gill 2003) (Figs. 15.7a, b and 15.8).

Implants manufactured according to the SLM process yield densities of approximately 100% without postprocessing steps. Postprocessing procedures, however, are necessary to stabilize titanium molecules of

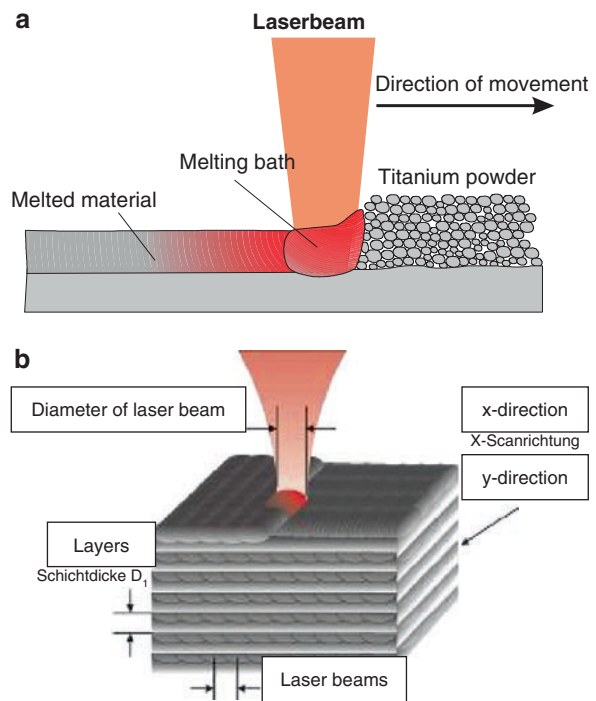


Fig. 15.7 (a) Manufacture of SLM implants: melting titanium powder. (b) Manufacture of SLM implants: layered technique

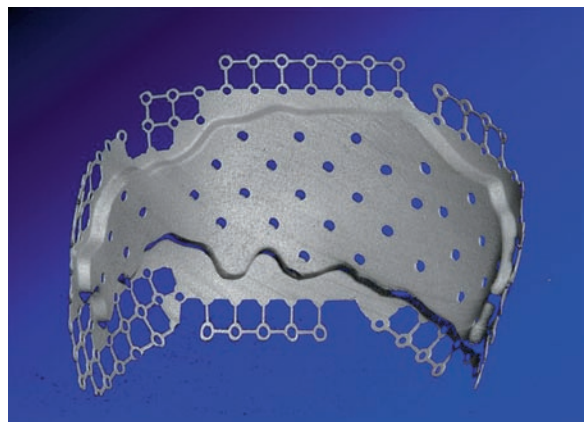


Fig. 15.8 SLM manufacture of a cranial implant with hollow and grid structures

the outer layers to prevent them from dissolving after implantation.

Acidic dipping followed by heating stabilizes the molecular structure of the implant. The characteristic of titanium enables its design and manufacture in complex three-dimensional forms. The main advantages of the SLM process are:

- Manufacture of complex geometries
- Direct rapid production of customized geometries

- Additive manufacture without loss of unprocessed material
- Fabrication of:
 - Defined roughness
 - Graded porosity
 - Lattice structures for realization of adapted stiffness

In addition to the medical aspects, the economic aspects are of great importance for custom made implants. The construction and manufacture of individual implants for craniofacial application are time-consuming and costly. The cost-dominating factors are the expenses for design, construction, and manufacturing, particularly for large defects. Design and manufacturing comprise several processing steps:

- Tooling
- Forging
- Casting
- Forming
- Machining
- Finishing

Costs are mainly determined by speed and effort of design and manufacturing, and both processes need an exact tuning and optimization in order to meet the demands of the surgeons. In comparison with conventional implant manufacturing, the production time needed for an SLM implant can be reduced by about 50%.

Potential advantages are reduced operating costs due to shorter operation times and shorter hospitalization.

Considering the overall objective of specialized medical centers, an implementation and certification of an effective and integrated process chain to plan and manufacture customized implants within 48 h seems to be possible today (Fig. 15.9). On the basis of the SLM process, new designs and material applications will become possible. Porous surfaces with a drastic reduction of the metallic content of the implant by creating three-dimensional spongy structures will allow material compositions with biomaterials without reduction in stability and precision.

15.3.2 PEEK-Implants

Polyetheretherketone (PEEK) is a semi crystalline thermoplastic polymer with biophysical properties similar to that of human bone (Jahur-Grodzinski 1999). The physical characteristics of the material combine good strength and stiffness similar to that of bone with excellent thermal and chemical properties. Its excellent biocompatibility makes this material ideal for long-term medical implant applications. With a continuous use temperature of 260°C, this polymer is suitable for every clinical sterilization method. The manufacture of

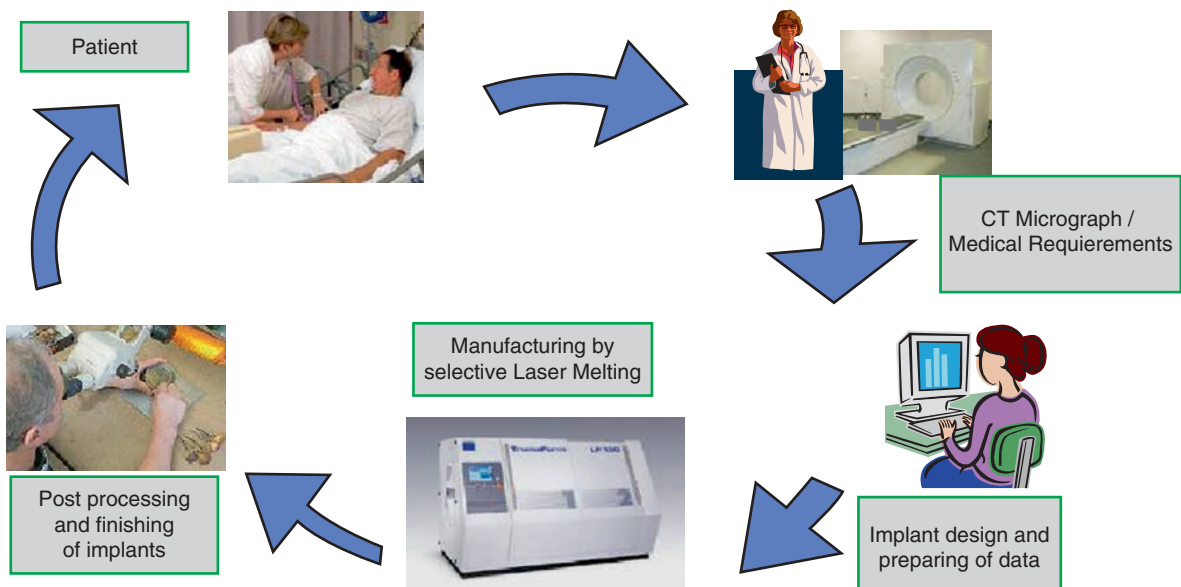


Fig. 15.9 Process chain for individual implant manufacture

individually designed implants can be performed in different ways. A production process as described above is possible, based on laser melting/sintering techniques from individual computer data (Rechtenwald et al. 2004; Schmidt et al. 2007).

PEEK is a fine powder and commercially available. Materials with low melting viscosity and a homogeneous distribution of particle size are best suited for the laser sintering process. The average particle size varies between 50 and 150 μm with irregular, edged particles. The irregular particle size makes it necessary to sieve the material before laser sintering as layers of 100–150 μm seem to be ideal for the layer-by-layer sintering technique.

To improve the biological and physical properties of the material, other allogenic and xenogenic materials can be added. Addition of nano-sized carbon black improves the flow characteristics of PEEK for modeling the implant. Organic and inorganic biological materials can be added to improve the biological response of tissues on nondegradable polymers as in-vivo application of PEEK results in an encapsulation of the implant with fibrous tissues, isolating the material from the surrounding bone (Balani et al. 2007). It has been shown that addition of bioactive ceramics such as Bioglass and sintered hydroxylapatite enhances osteoblast proliferation. In-vivo experiments with biologically altered PEEK basis material resulted in bony ingrowths into the implants based on a bone-like apatite layer on the surface of the implant (Rodil et al. 2005; von Wilmsowsky et al. 2008).

PEEK combines the excellent manufacturing properties of SLM-titanium implants with the advantages of an outstanding material with chemical and physical properties with a close resemblance to bone. The general problem of PEEK materials is fibrous encapsulation. As promising studies have shown, this might be overcome in the near future. Therefore, laser-sintered PEEK implants appear to be ideal as bone substitutes for various indications.

15.3.3 Outlook

Based on the experience with complex three-dimensional data sets, the development of implants with completely new features is close at hand. The features are:

- Well-defined porosity for improved bone ingrowth
- Adapted stiffness and elasticity close to that of bone
- Maximum reduction of allogenic material

The realization of these features requires the following work items:

- Dimensional accuracy $\leq 0.1 \text{ mm}$
- Fabrication of thin lattice structures with a detail resolution of 150–200 μm , made of titanium or polymers
- Smooth transfer of biomechanical loads from the natural bone into the metallic/polymeric implant based on Finite Element Method (FEM) implant design
- Fabrication of graded surface porosity
- Fabrication of surfaces with defined roughness ($R_z = 15 \text{ }\mu\text{m}$ up to 100 μm)
- Implant manufacture for all kinds of bone defects

The SLM technique, based on titanium and PEEK polymeric materials, is available for clinical use today. However, as described before, the technical process of transforming a virtual three-dimensional data set into real existing implants is challenging. The process chain demands a close cooperation between IT engineers, the manufacturing team and surgeons to guarantee a successful reconstruction. Today, this ideal setting is only available in a few medical centers.

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